Clinical Orthopaedics

ANTHONY F. DePALMA

Editor-in-Chief

With the Assistance of the
ASSOCIATE EDITORS
THE BOARD OF ADVISORY EDITORS
THE BOARD OF CORRESPONDING EDITORS

Number Seven

PITMAN MEDICAL
45 NEW OXFORD STREET
LONDON, W.C.1.

J. B. LIPPINCOTT COMPANY
Philadelphia and Montreal

Distributed in Great Britain by Pitman Medical Publishing Co., Limited, London

Library of Congress Catalog Card Number 53-7647

Clinical Orthopaedics is designed for the publication of original articles offering significant contributions to the advancement of surgical knowledge.

Original, typed manuscripts, not carbon copies, and illustrations should be forwarded prepaid to Dr. Anthony F. DePalma, 1025 Walnut Street, Philadelphia 7, Pa.

Manuscripts should be typed double spaced on one side of standard typewriter paper, leaving wide margins While every effort will be made to guard

Dictionary (edition 22) and Webster's *New International Dictionary* (edition 2) should be used as standard references. Scientific names for drugs should be used when possible. Copyright or trade names of drugs should be capitalized. Units of measurement, e g., dosage, should be expressed in the metric system Temperature should be expressed in degrees centigrade. A contribution in a foreign language, when accepted, will be translated and published in English

Black-and-white illustrations will be reproduced free of charge, but the publisher reserves the right to establish a reasonable limit upon the number. Ordinarily, colored illustrations cannot be published *except at the author's expense.* Black-and-white photographs should be in the form of glossy prints. *These should not be defaced in any way* Any changes desired in them should be marked on a tissue overlay This should be done before it is pasted to the print, since it is important not to mar the print in any way. Or any changes may be indicated on a separate sheet of paper Line and wash drawings should be on white art board, with lettering in black India ink large enough to be readable after necessary reduction. Large or bulky illustrations should be accompanied by smaller glossy reproductions of the same to facilitate their circulation among the members of the editorial board. Illustrations should be numbered, the tops indicated, and the author's name and the title of the article in brief should appear on the back. *However, this should be done lightly, so as to leave no imprint on the face of the illustration.* A separate typewritten sheet of legends for the illus-

Quarterly Cumulative Index Medicus:

If a book·

Author's name, title of book, edition if there is more than one, page numbers if it is wished to direct the reader to a specific section of the book, publisher's name, city, year of publication of book, in the order named.

If an article in a journal:

Author's name, title of article, volume number, inclusive page numbers, year of publication, in the order named.

Manuscript may be submitted to us in the original language of the author. Now it is our policy to handle the translation of these articles by our office without cost to the contributor if the article is found to be acceptable for publication

All manuscripts should be submitted with an extra carbon copy, including a short synopsis of approximately 200 to 250 words for translation into Interlingua.

Following are the general subjects of forthcoming issues of *Clinical Orthopaedics*

Chronic Hereditary Diseases and Developmental Anomalies, Fall, 1956
The Pathologic Physiology of Metabolic Bone Diseases, Spring, 1957
Affections of Growth Centers (Epiphyses, Apophyses), Fall, 1957
Orthopaedic Surgery in the Geriatric Patient, Spring, 1958
Rehabilitation, Fall, 1958

All contributors desiring to submit articles for consideration for publication on the topics listed above or in the general sections of this publication should submit them to the editor some months in advance of the date of the issue for which they are intended

PRINTED IN THE UNITED STATES OF AMERICA

Contents

Section I

TUMORS OF BONE

Section II
GENERAL ORTHOPAEDICS

Section III
MOTORIST INJURIES AND MOTORIST SAFETY
Part 1 Clinical Aspects

Guest Editor: Jacob Kulowski, M.D.
Saint Joseph, Missouri

CORRECTION

Clinical Orthopaedics 6. We regret that in Chapter 5 we omitted inadvertently to acknowledge the source of Figure 1. This was taken from an article by Dr. William J. Tobin in the *Journal of Bone & Joint Surgery* of January 1, 1956.

Section I

TUMORS OF BONE

1

Joseph Colt Bloodgood

Biographical Sketch

CHARLES F. GESCHICKTER, M.D.*

Joseph Colt Bloodgood, who rightfully can be called one of the chief founders of surgical pathology in America and the first consistent advocate of the use of frozen section biopsy in surgical diagnosis, was born in Milwaukee, Wisconsin, on November 1, 1867. He was the son of Francis and Josephine Colt Bloodgood and 1 of 4 children.

Dr Bloodgood decided early on a medical career, and, after receiving his Bachelor of Science degree in 1888 in his home state at the University of Wisconsin, he enrolled as a medical student at the University of Pennsylvania, where he received the M.D. degree in 1891, at the age of 24. In his later years Dr. Bloodgood referred frequently to the clinics that he helped prepare for Dr. Pepper. He recalled riding about in Dr. Pepper's carriage on house calls and being instructed to have a case ready for clinic in the morning. Dr. Pepper chose the subject for the clinic whether or not there was a case on the wards. Once he requested Dr. Bloodgood to prepare an empyema for presentation and, when Bloodgood remarked that there was no such case in the house, he replied irritably, "What difference does that make? Show any case and I will make it look like empyema " Dr. Bloodgood was one of the outstanding students of the class and was allowed to give night courses in embryology to his fellow students. Thus, his interest in teaching, which was to be one of his major activities throughout his professional life, was manifested prior to his graduation from medical school.

On graduating from medical school, Dr. Bloodgood interned in surgery at the University of Pennsylvania. He was caught in the fire of enthusiasm about the opening of the new hospital in Baltimore that had been so richly endowed by Johns Hopkins, and, through the influence of Dr. William Osler, also of the same faculty, he left to follow his career as a surgeon in Baltimore the following year.

Dr. Bloodgood joined the surgical staff of Dr. William Stewart Halsted at Johns Hopkins Hospital in 1892, and, after visiting the surgical clinics in Europe, became chief resident surgeon from 1893 to 1897. He was succeeded in this position by Dr. Harvey Cushing. On December 1, 1908, at the age of 41, Dr. Bloodgood married. Therefore, during his entire residency at the hospital, he was a bachelor and devoted his full time to his hospital duties and co-operated with great zeal and energy in the investigative studies carried out by both Dr. Halsted, professor of surgery, and Dr. Osler, professor of medicine. It is interesting that both Halsted and Osler married later in life—Halsted at the age of 38, Osler at the age of 43. Since Dr. William H. Welch, professor of pathology, was a bachelor until his death at

---

* Professor of Pathology, Georgetown University Medical School, Washington, D. C.



Joseph Colt Bloodgood

the age of 85, the resident quarters of the hospital staff was both a bachelor's club and a medical fraternity. Dr. J. M. T. Finney, who was a contemporary and colleague on the hospital staff, never received the residency because of his close family ties and frequent trips to the homestead at Belair, Md. In Halsted's own words, "We wish men who have learned to work for work's sake, who find in it and in the search for truth their greatest reward." Even after Halsted's marriage he spent months of bachelorhood in Baltimore during the summer, since Mrs. Halsted left in the spring for the family seat in High Hampton, North Carolina, and did not return until late fall.

Shortly after Bloodgood's appointment as resident, at the suggestion of Dr. Halsted, he undertook the study of the pathologic anatomy of all tumors and other tissues removed at operation. For this purpose, Halsted had assigned to him the southeast room on the second floor of the old pathology laboratory across the hall from the room of Dr. Welch, a room that later was used also by Dr. Walter Reed. Later, Dr. Bloodgood was assigned the large front room on the third floor of the new surgical building, where the first operation was performed by Dr. Halsted in 1904. Both of these buildings, in which Bloodgood did his early work in surgical pathology, have since been demolished to make room for new structures. Thus, Dr. Bloodgood began his studies in surgical pathology while still a surgical resident, and after a few years he began courses in surgical pathology for the medical students. These lectures and studies he pursued with unending interest until the time of his death.

In 1899, two years after completing his surgical residency, Dr. Bloodgood reviewed the results of 459 cases of Dr. Halsted's radical operation for inguinal hernia. It was Dr. Bloodgood who analyzed also the records of Dr. Halsted's radical operation for cancer of the breast. Halsted's first report was in 1894, and it included 50 cases. In the words of Dr. W. G. MacCallum, these studies of Bloodgood "served as a solid foundation for so many of Dr. Halsted's own contributions." In his article reporting the result of operation on 232 cases of breast cancer in 1907, Halsted wrote: "It affords me the greatest pleasure to express anew my obligations to Dr. Bloodgood for his efficiency and inexhaustible zeal in collating facts year after year for so many years."

Throughout Dr. Bloodgood's busy life as a practicing surgeon and a teacher, his intense interest in the follow-up studies on the results of treatment of tumors was one of the most amazing aspects of his career. In 1910, he organized a fund for follow-up studies and cancer research under his own name, and, although in later years his laboratory contained the records of nearly 30,000 tumors, he insisted that both the physician and the patient receive annual or semi-

annual letters. Throughout the years "The patient is reported living and well" was a byword in his laboratory, and in his pathology lectures he loved to add the comment, "The patient is living, but the doctor died." When one considers the arbitrary classification, reclassification and subclassification that has confused the issue in the field of neoplasms and the numerous attempts at naming and renaming identical lesions in order to get a new catch phrase in the literature, it is amazing to recall that here was a man who believed in characterizing tumors on the basis of their biologic behavior as determined by adequate follow-up studies. In the last analysis, it is how a tumor behaves in the body of the patient, whether treated or untreated, that determines its significance for the profession and for the living host. Terminology is an accessory after the fact and could be substituted by numerical typing. Maybe this is the reason why today no tumor bears Bloodgood's name, although he was the first to coin the phrase "benign giant-cell tumor," in spite of the fact that his colleague in general pathology, Dr. MacCallum, insisted on calling them giant-cell sarcomas to the end. He also popularized the benign blue dome cyst of the breast, although Sir Astley Cooper had described accurately the blue dome nearly 75 years previously.

Bloodgood's major contributions were in the study of tumors of bone and tumors of the breast. He published about 60 articles on bone and approximately 80 on lesions of the breast; the best of these articles were contributed between 1920 and 1925 after his return from the Army, in which he was a major in World War I.

The foundation for Dr. Bloodgood's later articles on bone tumors was laid by years of a thorough review of the literature. These reviews were reported between 1910 and 1919 in *Progressive Medicine,* published by J. B. Lippincott Company. The heading of the reviews is interesting. Between 1901 and 1905 the title of the review was "Anesthesia, Fractures, Dislocations and Amputations." Between 1906 and 1909, the topics covered were the same, but they were extended to include Shock and Surgery of the Joints, and between 1913 and 1918 there was added to the above title the word *tumors.* These comprehensive reviews were contributed faithfully over a period of 18 years and were discontinued when Dr. Bloodgood entered the Army in World War I. They never were resumed because, in Dr. Bloodgood's own words, they were too time consuming as his practice grew. Moreover, after the war many of the former residents at Hopkins, instead of going back to their previous assignments, were encouraged to remain in Bloodgood's laboratory for a year to assist him in laying down a "creeping barrage" of contributions on tumors of bone and breast. The war, therefore, was responsible for changing Bloodgood's emphasis in his publications and also responsible for the large number of articles that appeared under his name between 1920 and 1925. During the 18-year period when Dr. Bloodgood was reviewing the literature for *Progressive Medicine,* the articles in French and German, as well as in English, were followed assiduously. Dr. Bloodgood was not a great linguist, and his work would have been impossible without the aid of Herman Shapiro, who did the translations. Mr. Shapiro entered Bloodgood's service on the recommendation of Dr. Halsted, who had employed him previously for similar purposes. So, in essence, both Halsted and Shapiro were co-authors. In Bloodgood's own words, it was from Halsted that "I learned the names of all the German surgeons connected with German universities and about their work. He also familiarized me with the great clinic in Vienna." Mr. Shapiro told the author that when Dr. Bloodgood's interest in reviewing the literature lagged, he would make him close the laboratory door, sit at his desk and make notes while Shapiro put into English, line by line, the articles in German and French that he had gathered from the library.



Joseph Colt Bloodgood

the age of 85, the resident quarters of the hospital staff was both a bachelor's club and a medical fraternity. Dr. J. M. T. Finney, who was a contemporary and colleague on the hospital staff, never received the residency because of his close family ties and frequent trips to the homestead at Belair, Md. In Halsted's own words, "We wish men who have learned to work for work's sake, who find in it and in the search for truth their greatest reward." Even after Halsted's marriage he spent months of bachelorhood in Baltimore during the summer, since Mrs. Halsted left in the spring for the family seat in High Hampton, North Carolina, and did not return until late fall.

Shortly after Bloodgood's appointment as resident, at the suggestion of Dr. Halsted, he undertook the study of the pathologic anatomy of all tumors and other tissues removed at operation. For this purpose, Halsted had assigned to him the southeast room on the second floor of the old pathology laboratory across the hall from the room of Dr. Welch, a room that later was used also by Dr. Walter Reed. Later, Dr. Bloodgood was assigned the large front room on the third floor of the new surgical building, where the first operation was performed by Dr. Halsted in 1904. Both of these buildings, in which Bloodgood did his early work in surgical pathology, have since been demolished to make room for new structures. Thus, Dr. Bloodgood began his studies in surgical pathology while still a surgical resident, and after a few years he began courses in surgical pathology for the medical students. These lectures and studies he pursued with unending interest until the time of his death.

In 1899, two years after completing his surgical residency, Dr. Bloodgood reviewed the results of 459 cases of Dr. Halsted's radical operation for inguinal hernia. It was Dr. Bloodgood who analyzed also the records of Dr. Halsted's radical operation for cancer of the breast. Halsted's first report was in 1894, and it included 50 cases. In the words of Dr. W. G. MacCallum, these studies of Bloodgood "served as a solid foundation for so many of Dr. Halsted's own contributions." In his article reporting the result of operation on 232 cases of breast cancer in 1907, Halsted wrote: "It affords me the greatest pleasure to express anew my obligations to Dr. Bloodgood for his efficiency and inexhaustible zeal in collating facts year after year for so many years."

Throughout Dr. Bloodgood's busy life as a practicing surgeon and a teacher, his intense interest in the follow-up studies on the results of treatment of tumors was one of the most amazing aspects of his career. In 1910, he organized a fund for follow-up studies and cancer research under his own name, and, although in later years his laboratory contained the records of nearly 30,000 tumors, he insisted that both the physician and the patient receive annual or semi-

back on this amazing performance and his teaching activities, it appears that Dr. Bloodgood was more interested in pathology than in surgery.

It was his custom to have his entire force meet him in the laboratory on Sunday mornings. They would go over the histories and the specimens of the cases that he was reporting in the literature. At his direction, Edward Walker (his laboratory technician) would cut dozens of frozen sections from the formalin-fixed specimens, and the slides of the original operation would be reviewed. Representative areas would be photographed by his photographer Herman Shapiro, and he would dictate follow-up letters to the patient and the referring physician. These sessions often would last from 10 A.M. to 4 P.M. without time out for lunch.

On Sunday nights he dictated articles at home to Herman Shapiro—who also was a court reporter—to whom he usually referred as his librarian. These articles were based on his own material, his previous articles, and they never contained a bibliography. Mrs. Bloodgood sat by quietly mending socks. One of us had to be present with the histories and the tabulations from the laboratory records. In the summer this procedure went on at Gibson Island, 25 miles from Baltimore. I never knew a time when he did not have several suitcases full of case histories at his home, and they were being returned and replaced by others repeatedly.

He frequently took advantage of Mrs. Bloodgood's absence from their residence at 44 Warrenton Road to spread the histories out with their lantern slides and photographs on the dining room table. We would work ... ously over the week end. The serv... s would be dismissed early. We would ... the meals, and Dr. Bloodgood would ... wash the dishes. During the entire time ... versation never veered from the sub... study. Tumors of breast, bone, ... oral cavity were the favorite

... d never refused an invitation to talk at a medical meeting. If possible, he preferred to be driven out of town to these medical clinics, even though there might be miles of detour on the road. He carried 2 large suitcases fully packed, and he was prepared equally to spend an evening or an entire week.

I did not know Dr. Bloodgood in the earlier years when (like other "Hopkins prima donnas") he was said to have been a tyrant in the operating room and in the laboratory. This was certainly true of his other colleagues—Cushing, Dandy, Crowe and others. However, in the 10 years that I worked with him, I never heard him utter a harsh or a profane word. He never had an unkind word to say about anyone, and his motto was, "If there is nothing good to be said—say nothing."

When Dr. Murray M. Copeland and I were working on bone tumors in his laboratory, we stayed as guests and sole occupants of his residence the entire summer. His car, a Ford, was given over to us, and servants and food went along with the establishment. We were supposed to keep the grass cut, but this already had been arranged for through professional gardeners.

In the summer when he went to Europe, his summer home was at our disposal. Here the entire laboratory force was entertained every week end throughout the entire summer.

Because of his traditional generosity, his office tried to keep him without pocket cash, but this did not prevent him from using his account at the Maryland Club in Baltimore and the Athletic Club in New York for the benefit of his own force and the resident staff of numerous hospitals.

Dr. Bloodgood believed that his greatest contribution in tumors was his observation that cancer never begins as cancer but in a focus of abnormal tissue, the site of a previous benign change. Therefore, not only was cancer curable in its early stages, but if the preceding abnormality could be recognized and treated, it could be prevented.

Dr. Bloodgood was a tedious operator at times, although he could operate speedily and skillfully with the best. However, this was not his ordinary custom. Unlike most surgeons, he was prone to stop during an operation, and, after removing numerous tissues for frozen section biopsy, it was his habit to drop out of the operation temporarily and look at the frozen section under the microscope.

Dr. Bloodgood loved to put on a startling demonstration at medical clinics or in the operating room. He preferred to work before an audience. In the late twenties and the early thirties, when the staff in the Surgical Pathology Laboratory had been expanded, he put on 4 postgraduate demonstrations yearly, each lasting 3 or 4 days with morning, afternoon and evening sessions. The initial one was a lantern slide demonstration of bone tumors; the remainder, microscopic diagnostic exercises with sections of various tumors. The medical audience participated by voting on the diagnosis and, at times, on therapy to be recommended. This was the beginning of the diagnostic microscopic exercises now conducted annually by the American Society of Clinical Pathologists. He enjoyed using 4 lantern slides simultaneously, illustrating a single case with 4 lanterns, 4 screens and 4 operators. Slide 1 was the picture of the patient; slide 2, the roentgenogram; slide 3, the gross specimen; and slide 4, the microphotograph.

In the operating room he often had working with him 2 assistant surgeons, Dr. George A. Stewart and Dr. L. Clarence Cohn, as well as 3 or 4 interns, all scrubbed and all participating simultaneously in the procedure In addition, an unscrubbed frozen section team often danced attendance on the chief operator, weaving in and out of the operating room In the middle of a major operation he might drop out and transfer his activities to another operation proceeding simultaneously in another room. His clinics often were referred to as a 3-ring circus. Like a quarterback who baffles the opposition, he was forever changing his operating room tactics. When his staff was prepared to have him amputate with a tourniquet, he would decide suddenly to operate without one, tying each vessel separately as he proceeded. When they expected him to use a cautery, he would proceed without one. When they expected him to use the electric cautery, he would switch to plumber's soldering irons.

He once asked Dr. Seegar, chief gynecologist at the St. Agnes Hospital, to remove a right ovarian tumor while he was removing the left, and no sooner had Dr. Seegar's team started to assist than he covered their operating field with numerous Mikulicz pads wrung out of warm saline. Once he telephoned the resident surgeon at the hospital at midnight to inform him that he was sending in an acute gallbladder case by ambulance and requested him to meet the patient at the door with a stretcher. When the resident had the temerity to ask which door, he replied, "Have a stretcher at every door." (There were 4 entrances!)

In the later decades of his life, practically all his surgery was performed at St. Agnes Hospital, and his office was maintained at 904 N. Charles Street, Baltimore, Md., except for 3 years at 3301 North Charles Street.

He dictated elaborate operative notes to the secretary at St. Agnes and telephoned equally detailed descriptions to the secretary at Johns Hopkins Hospital immediately after each operation He demanded that 5 copies be made of every report, 1 to be filed at the hospital and 1 at the office, another to be sent to the referring physician, and the 2 remaining copies to be kept on file at the Surgical Pathology Laboratory at Johns Hopkins Hospital Each report carried the hospital number, his office number (known as the JCB number) and the pathology number. In addition, duplicate copies were kept of all correspondence. Thus, between 25,000 and 30,000 case reports were kept, all of them filed in the laboratory and classified by organ and diagnosis. In looking

2

Tumors of Cartilaginous Origin

MURRAY M. COPELAND, M.D., F.A.C.S.*

Embryonic processes concerned in the formation of bone are important factors in determining the nature of bone tumors that may arise. It is now established that development of the human skeleton does not lead to a definitive form in early adulthood, and that there persist developmental foci distributed in accordance with prearranged structural patterns. These developmental foci carry with them a neoplastic liability.

The majority of benign and malignant lesions of bone occur in endochondral bone during adolescence, the period of greatest bone growth activity, at or medial to the growth zone Embryonal processes occurring in this zone include: orderly arrangement of blastemal tissue into the pattern of subsequent skeletal structure (Fig. 1); cartilaginous growth; calcification of cartilaginous intercellular substance; the resorption and the vascularization of cartilaginous tissue leaving calcified spicules to act as a base for endosteal osteoblastic activity; ossification of endosteal spicules and periosteal fibrous tissue (Fig. 2); subsequent growth of cortex and revascularization of early endosteal bone leading to re-formation of more permanent and useful endosteal bone.

Primary tumors of cartilaginous structure and origin arise from persisting islands of cartilage that may be found within the medullary substance; in the subperiosteal tissue at or near the attachment of tendons to tuberosities; or in the periarticular regions. Skeletal embryonal tissue may persist as such at any of these locations, as well as within the synovium of the joint. It may be recognized histologically as small compact spindle cells or stellate cells. This tissue, when undergoing tumor formation, may reveal both cartilage and bone with very little resorptive phase.

Cartilaginous tumors of periosteal origin may be either benign or malignant. The benign lesions, such as osteochondroma, appear principally at the sites of insertion of certain tendons, such as the adductor magnus muscle tendon insertion; the quadriceps tendon attachment to the medial aspect of the upper tibia; the deltoid tuberosity; and the region of the femoral trochanters. Such foci, when undergoing neoplastic change, displace normal blastemal tissue transition, with accelerated growth but without distortion of tissue differentiation (connective tissue, cartilage and bone). When primary malignant transformation (primary chondrosarcoma) occurs at the same sites, cellular connective tissue and islands of calcification predominate. It would appear from anatomic, pathologic and clinical studies that the tissue of origin is the same for both the benign and the malignant cartilaginous lesions.

Persisting islands of cartilage found in the medullary substance of various bones in the body may remain quiescent or undergo subsequent growth, giving rise to central chondromas. Such lesions are seen more commonly in the bones of the hand, the foot, the sternum, the spine and the pelvis, though central chondromas occasionally are seen in

* Department of Oncology, Georgetown University Medical Center, Washington 7, D. C.

Therefore, cancer was a preventable disease. This message he wanted to strike home to both the medical profession and the public, and he was not averse to enlisting the aid of newspapers in his crusade. For this he was criticized severely by some of his younger surgical confreres in Baltimore, who neither knew the man personally nor appreciated his purpose. Some of them tried unsuccessfully to have him expelled from the local medical and surgical society (Medical and Chirurgical Faculty of Baltimore) on the grounds that thus he was attempting to enlarge his private practice.

This schism in the Medical and Chirurgical Faculty of Baltimore was in some measure the result of the peculiar character of Bloodgood's publications between the years 1930 and 1935. While he still continued to write original contributions for such societies as the American Surgical Association, the majority of his contributions were hardly of professional medical character, and many of them were written for dentists, nurses and the public at large. Particularly astounding was his late interest in the prevention of cancer in the cervix of mothers by providing correct information to the laity. As early as 1921 and 1922, Bloodgood had published a series of articles under the title of "Cancer of the Tongue (Oral Cavity) a Preventable Disease," but he had never before been a contributor to gynecologic literature. His advocation of periodic examinations of apparently normal individuals for the detection of precancerous lesions was a pioneer undertaking, aimed at both the medical profession and the public. Today, routine examination to rule out the presence of cancer is a recognized procedure in all the major hospitals of the United States and is supported by the American Cancer Society. But no one ever pauses to recall the heartaches of the pioneer who was regarded as a sheep gone astray in his attempt to popularize the idea.

Besides Dr. Howard A. Kelly, he was perhaps the only surgeon of the Hopkins School who had any concept of the value of irradiation in cancer. The harsh treatment that both Dr. Kelly and Dr. Bloodgood received in their latter years at the Hopkins was probably the darkest blemish on the otherwise shining emblem of this truly great institute of medical learning.

Dr. Bloodgood with his robust physique, his contagious smile and his inbred qualities of a true gentleman, died at home on October 22, 1935, at the age of 67 of his second attack of myocardial failure.

In the evaluation of his career, his role as surgeon and operator stands last in the list of his accomplishments. He is remembered primarily as a surgical pathologist who brought the microscope into the operating room and, through the instrument of the frozen section, enabled numerous surgeons to spare countless legs, arms, breasts and other organs from mutilation. In the literature of tumors of bone and tumors of the breast he will be remembered for his interpretation of giant-cell tumors and chronic cystic mastitis as benign lesions that can be handled by conservative measures. In the crusade against cancer he will be known for advocating the importance of the follow-up study as a technic for the evaluation of methods of diagnosis and treatment; and in the attempt to prevent the disease he will be recognized as a pioneer in advocating periodic examinations for the disclosure of unsuspected tumors and precancerous lesions and as stressing the importance of public education in this regard

tissue appears to be responsible for chondromatosis of the joint and probably some of the osteophytes found in arthritis.

In the adolescent period there is at the epiphyseal lines, such as in the upper end of the humerus, the lower end of the femur, etc., a final accentuation of growth. This growth takes place on the metaphyseal side of the epiphyseal line by reproduction of cartilage cells in the form of chondroblastic tissue, with subsequent maturation and development of the cartilage cells. This is followed by vascularization with resorption of the calcified material and with replacement by permanent new bone. The cartilage cells seem to play no active role in this final ossification. Chondroblastomas, benign and malignant, may supervene in these areas. The chondroblastic new growth coincides with the normal process of development but persists as chondroblastic tissue, ending in calcified cartilage without producing the usual matrix. The vascularity and giant-cell invasion are secondary features. Malignant transformation may occur.

The bone tumors of cartilaginous origin related to the phases of skeletal development are as follows: (1) cartilaginous growth is represented by central chondroma, chondrosarcoma, hereditary chondrodysplasia and chondromatosis of joints; (2) cartilaginous growth, followed by vascularization, resorptive changes and subsequent ossification, is reflected in the development of osteochondromas, chondroblastomas and sarcomas of mixed osteogenic and cartilaginous origin.

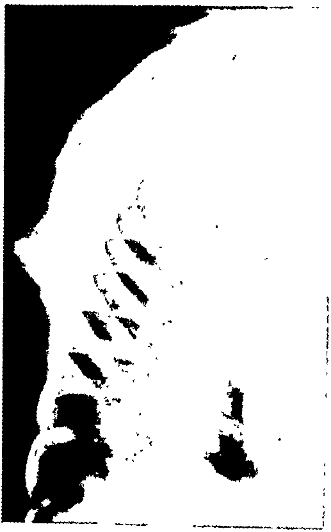


FIG. 3. An immature cartilaginous exostosis situated in the left scapula of a young child. The large translucent defect is consistent with a benign lesion, but it must be differentiated from chondrosarcoma or osteolytic osteogenic sarcoma. (Dr. William Tobin)

TABLE 1. CLASSIFICATION OF CARTILAGINOUS TUMORS*

1. Osteochondroma (solitary and multiple)
2. Chondroma or chondromyxoma (solitary and multiple)
3. Chondroblastoma, benign and malignant
4. Chondromatosis of joints
5. Chondrosarcoma, primary and secondary

* After Geschickter and Copeland: Tumors of Bone, ed. 3, Philadelphia, Lippincott, 1949.

OSTEOCHONDROMA

This is the most common benign tumor of bone. The lesions are found most commonly near the ends of long bones of patients between 10 and 25 years of age. Nearly one third of the lesions are found about the knee. The other sites of importance are the shoulder girdle, the bones of the thoracic cage and the small bones of the hands and

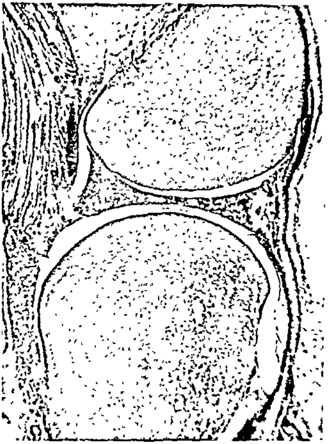


FIG 1. Human embryo, approximately 100 mm, showing embryonic connective tissue forming the preliminary structure of the femur condyle and patella. Note the embryonic connective tissue present in the joint cavity undergoing regression.



FIG. 2. Cortex of a femur from fetus, showing the beginning rim of osteoid structure about the margin of the skeletal blastemal tissue. Note the development of the haversian canals and the fetal periosteum.

the long bones. It is interesting to note that many of the common sites are in the regions of multiple joints

Multiple osteochondromas and central chondromas probably have similar backgrounds of persistent cartilaginous foci. The developmental disturbances of periosteum about tendinous attachments and a general lag in periosteal development produce a multiplicity of effects on the bone beneath. The cortex may not reach its full thickness; multiple outgrowths appear near the epiphyseal regions where growth is most rapid, with irregularity and widening of the metaphyseal ends of the bones.

Secondary chondrosarcoma can be traced to a pre-existing benign osteochondroma or central chondroma. Often, repeated surgical excision or trauma precedes the appearance of malignant transformation in the cartilaginous tissue The real difficulty in advanced lesions of this type is to determine whether the chondrosarcoma is of primary or of secondary origin. Central chondromas of the hands and the feet rarely give rise to malignant transformation, whereas the same cartilaginous tumors in the other bones of the body have a potentially serious prognosis.

Chondromatosis of the joints originates in the capsule and the synovial membrane Strands of primitive precartilaginous tissue, which form the joint cavities in the embryo by mucoid regression, normally remain after birth at the edges of the articulations Such

a bony protrusion during the period of maximal skeletal development. Growth observed in these tumors after 30 years of age usually indicates a secondary malignant process, a transformation to chondrosarcoma.

Simple excision usually suffices to cure the osteochondromas that produce pain or dysfunction. In a large series of cases studied, 5 per cent of osteochondromas showed malignant changes and required radical extirpation by resection or amputation of the part, when this was possible.

MULTIPLE OSTEOCHONDROMAS OR HEREDITARY DEFORMING CHONDRODYSPLASIA

This entity denotes a distinct form of dysplasia in which the multiple occurrence of osteochondromas in a single patient is accompanied by numerous other skeletal deformities. There may be bending and shortening of the bones, together with widening and irregularity of their metaphyseal portion, due to epiphysial and periosteal disturbances. The hereditary character of this process has been traced through as many as 4 or 5 generations. Transmission may be through either parent. Stocks and Barrington found that the male parent was responsible for the inheritance in 73 per cent and the mother in 27 per cent of such cases. The fundamental basis for the congenital disturbance is obscure, but there is definite evidence that there are deficiencies in the periosteum, a tendency for the perichondrium to persist, and this, together with proliferating precartilaginous connective tissue,

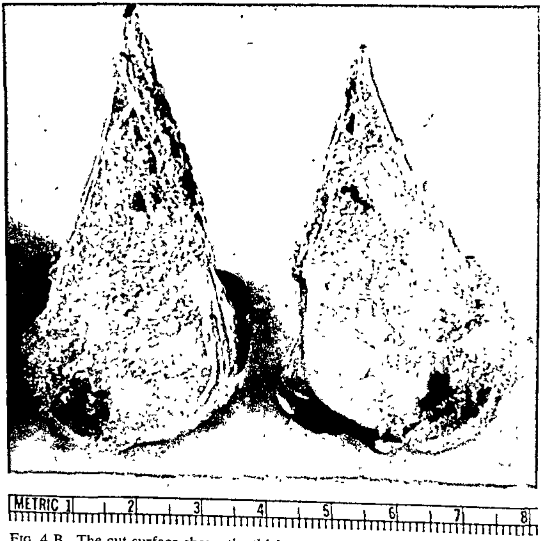


Fig. 4 B. The cut surface shows the thickness of the cartilaginous portion overlying the cancellous bone.

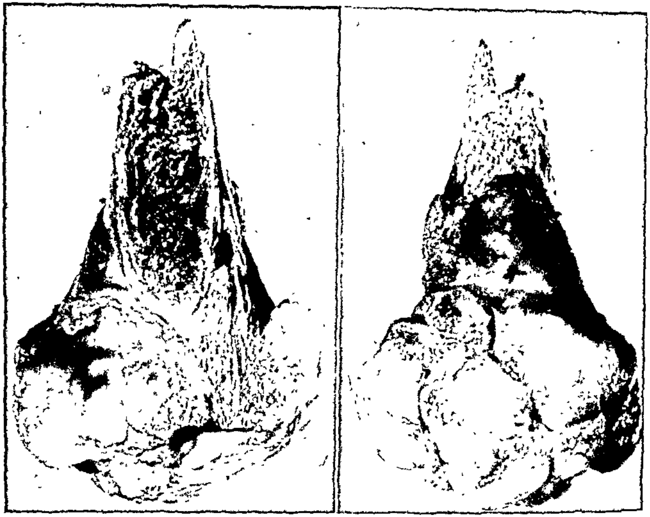


FIG. 4 A Gross specimen of an osteochondroma removed from the lower femur showing the cartilaginous cap composed of chondral tissue overlying the pedicle of bone.

the feet. The symptoms are of moderate severity and of long duration (average 5 years). Some of these tumors are of congenital origin (25%), with a familial history. Eight per cent of the cases are of the multiple hereditary variety. Acute symptoms may follow a sudden increase in growth. Edema, pain, deformity or dysfunction may bring the patient under observation.

The site of the lesion determines the readiness with which the lesion may be discovered. Physical examination yields little information unless deformity exists. Pelvic bone lesions are rarely discovered unless symptoms appear or an incidental roentgenogram is made.

The tumors are composed of a translucent cartilaginous cap, formed by proliferating precartilaginous connective tissue which also dips into the cartilage, dividing it into lobules (Fig. 5). Numerous areas of fetal cartilage may be present near the capsule, but the greater portion of chondral tissue is of the adult type (Figs. 3-5). Cancellous bone beneath the chondral tissue forms a base or a pedicle for the new growth Calcification within the cartilaginous cap is a frequent finding.

The roentgenogram of a typical benign lesion depicts compact differentiated outgrowth of bone and an overlying cartilaginous cauliflowerlike cap, flecked with calcium (Fig. 6).

These lesions may be left untreated, but they must be watched at intervals by roentgen examination. In growing children, roentgen pictures of such tumors indicate that they are taking origin in an abnormality of

CHONDROMA OR CHONDROMYXOMA (SOLITARY AND MULTIPLE)

This is a common type of cartilaginous tumor, composed predominantly of adult hyaline cartilage, centrally located, which replaces the normal cancellous and marrow tissue within the shaft of the bone. It occurs most frequently in the small bones of the hands and the feet, also in the spine, the ribs, the sternum and, occasionally, the long bones. It is seen most frequently in patients between 20 and 30 years of age. Gradual growth or pain following an injury is the most common history given by patients.

In the roentgenogram the tumor appears as a central radiolucent area, much like a cyst within a shell of cortical bone. Flecks of calcification or longitudinal trabeculae usually are noted within the cystic area (Fig. 8). Histologically, the tumor is composed of proliferating, precartilaginous connective tissue, which elaborates islands of adult hyaline cartilage (Fig. 9). The connective tissue strands dip between the cartilaginous islands and tend to form adult fibrous trabeculae in the more slowly growing tumors. In some chondromas, myxoma (precartilaginous connective tissue) may be present in varying amounts.

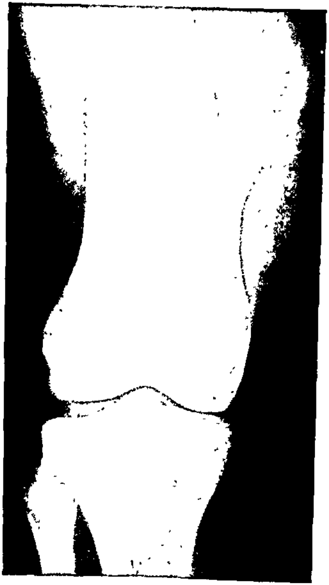
Lesions in the small bones of the hands and the feet may be looked upon as benign and curable by thorough extirpation (curettement and chemical cauterization). Only rarely has a central chondroma of the phalanges, the metacarpals or the metatarsal



FIG. 7. Roentgenogram of a case of hereditary chondrodysplasia. Note the clusters of outgrowths about the broad base of the femurs and the upper tibias.



FIG. 5. Photomicrograph of cartilaginous cap seen in Figure 4. At the outer margin are noted lobules of tumor separately overlying the cancellous bone is well cartilaginous type. The cartilage immediately overlying the cancellous bone is well developed and undergoing calcification. The peripheral cartilage shows features of immaturity. (After Geschickter & Copeland: Tumors of Bone, ed. 3, Philadelphia, Lippincott)



produces most of the deformities (Fig. 7).

The regions affected frequently and most severely are those of the forearm and the lower leg. The bones may be fused at one point. The prognosis, so far as life is concerned, is good; however, there is no adequate form of treatment except operation for correction of the deformities and removal of any osteochondroma showing growth or causing pain or dysfunction. Malignant degeneration occurs occasionally in this disease. In rare instances 2 or more of the lesions may show malignant change in the same patient. When necessary, resection or amputation is the treatment of choice.

FIG. 6. Osteochondroma of the pedicle type Note the widened metaphyseal region near the outgrowth and the extension of the pedicle in the direction of the pull of the adductor magnus muscle.

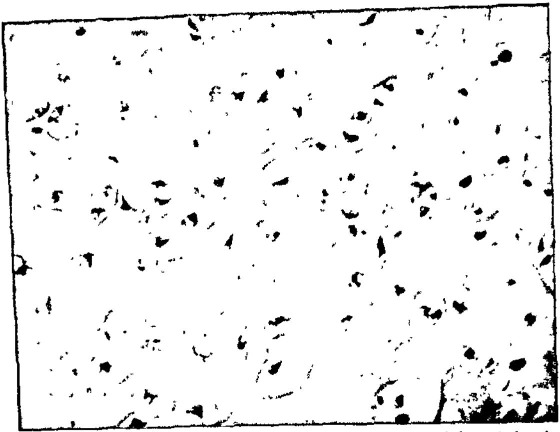


FIG 9. High-power microphotograph of cartilage removed from lesion in Figure 8, showing adult hyaline cartilage. There is beginning calcification of the matrix, which accounts for the calcific deposits noted in the roentgenogram.



FIG. 10. Roentgenogram of a benign chondroblastoma involving the epiphysial line of the lower end of the tibia and invading the metaphyseal region; evidence of periosteal reaction is seen. Note the defect in the lower shaft of tibia in the metaphyseal region, which represents an intracortical non-,ing fibroma of bone. (Dr. Austin Rohrbaugh)



FIG. 8. Roentgenogram of a central chondroma with a radiolucent area surrounded by flecks and longitudinal striations of calcification in the shaft of the humerus.

bones shown malignant propensities. True chondromas of large size occurring about the sternum or in the long bones must be looked upon as potentially malignant.

Occasionally, chondromas are multiple in distribution, and, when associated with hemangiomatosis, the condition is known as Maffucci's syndrome In multiple chondromatosis, the involvement of several small bones of the extremities is apt to be accompanied by a similar but larger defect near the end of one of the large bones. Deformities may appear early in life, but they become stationary in the early twenties. Trivial injuries may cause fractures through a chondromatous area. The patients usually present deformities.

When central chondromas are quiescent and have either mild or no symptoms, it is best to leave them alone and keep them under observation by roentgen examination, repeated annually. If they are subjected frequently to trauma because of their position, with resulting soreness and discomfort, their complete surgical removal should be undertaken. *This is especially true if the tumor is* in the usual location, i e., one of the small bones of the hand or the foot. In the phalanges, amputation may be necessary, but usually the lesion is small enough to permit preservation of bony continuity after thorough curetting, followed by sterilization with 50 per cent zinc chloride. The ease with which cartilaginous tumor tissue is transplanted surgically in the wound should be kept constantly in mind, particularly in the large chondromas. When recurrence of a cartilaginous tumor in a long bone takes place, resection or amputation is indicated. When a previously quiescent tumor shows sudden signs of increased growth, with an exacerbation of symptoms, immediate and complete removal is indicated, because secondary malignant change is to be expected In instances in which the tumor has grown to unusual size, or in which the location about the sternum or the spine makes complete removal difficult or dangerous, roentgenotherapy in the hands of a competent therapeutist may be tried. Due to improvement in surgical technic, conservative measures are not warranted except in unusual circumstances or because of the location of the tumor. Disarticulation and hemipelvectomy have established cures in cases of recurrent chondroma or chondrosarcoma in and about the pelvic girdle.



FIG. 9. High-power microphotograph of cartilage removed from lesion in Figure 8, showing adult hyaline cartilage. There is beginning calcification of the matrix, which accounts for the calcific deposits noted in the roentgenogram.



FIG. 10. Roentgenogram of a benign chondroblastoma involving the epiphysial line of the lower end of the tibia and invading the metaphyseal region; no evidence of periosteal reaction is seen. Note the defect in the lower shaft of the tibia in the metaphyseal region, which represents an intracortical nonossifying fibroma of bone. (Dr. Austin Rohrbaugh)

CHONDROBLASTOMA, BENIGN AND MALIGNANT

Chondroblastic tumors of bone arise from a proliferation of cartilage at the epiphyseal line at or near the age of puberty and are not frequent in their occurrence. Such tumors *of vascular and cartilaginous origin* usually are situated on the metaphyseal side of the epiphyseal line in long bones. They produce destruction by invading the cancellous spaces and exhibit an overlying periosteal reaction. The age limits are principally between 10 and 24 years. On the basis of age alone, this tumor may be linked with the process of bone growth where ossification, by way of temporary calcified cartilage, is taking place at the epiphyseal line during the period of adolescence. Localization of the tumor lends weight to this supposition.

The location of these tumors is largely in the ends of the long bones about the knee *joint (the upper tibia and the lower femur)* and in the upper humerus. The bulk of the tumor is on the shaft side of the epiphyseal line, though the epiphysis also is invaded in most cases.

There seems to be a correlation between the age and the duration of symptoms. In patients under 20 years of age, the average duration of symptoms is less than 5 months, while in an occasional patient of 30 or more years of age, the disease averages over 3 years. The duration of symptoms varies from 1 to 16 months. Pain, tenderness and swelling, followed by lameness in the affected member, are characteristic of the lesion as it occurs in weight-bearing bones. Occasionally, effusion in the knee joint suggests tuberculosis. Pathologic fracture has been a rare complication. Leukocytosis and significant enlargement of the regional lymph nodes are infrequent.

Roentgenographically, the lesions show rarefaction in the cancellous bone extending on either side of the epiphyseal line and, characteristically, with an overlying periosteal reaction along a single margin of the bone. Early stages of the growth are seen by

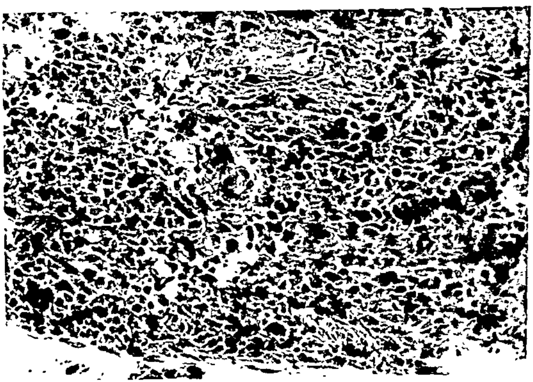


Fig. 11 Microscopic section from case shown in Figure 10, showing benign proliferation of chondroblasts with little evidence of calcified cartilage, osteoclasts are present

genogram primarily in the metaphysis, extending into the epiphysis in most (Fig. 10). While the tumor is pre- nantly central in location, the perios- usually shows a translucent shadow out much evidence of bone reaction. ıondroblastoma differs from benign gi- :ell tumor in that it involves both the hysis and the shaft of the bone; is not ined predominantly to the epiphysis; does expand beneath the shell of cortical bone; not have coarse trabeculae; and does a periosteal reaction. The age distribu- of benign giant-cell tumors and solitary of metastatic cancer practically always udes the years over 21, while chondro- ıtoma is found in younger age groups. asionally, Ewing's tumor may be a rce of confusion.

At operation, the tumor varies in con- ency according to the degree of vascu- ty and necrosis. Frequently it is indis- guishable from the hemorrhagic grumous terial found in giant-cell tumor, the hya- material in chondromas or tissue found so-called malignant aneurysms of bone. ıall cystic cavities may be noted in vari- s parts of the tumor.

There are 2 important objectives in the croscopic interpretation of chondroblastic mors: (1) to distinguish these growths om benign giant-cell tumors; (2) to sep- ate chondroblastic tumors into benign and alignant forms. The presence of chondro- asts and hyaline matrix, with or without e web of calcification traversing the hya- ıe material, differentiates these neoplasms om benign giant-cell tumors (Fig. 11). ivision of chondroblastic tumors into be- gn and malignant forms by microscopic udy is a more difficult task. In the benign owths, the nuclei of the chondroblasts are niformly small, the cytoplasm is moderate amount, stains faintly, and is ill defined its markings. The hyaline matrix is mod- rate in amount but infiltrates in fine strands ith well-defined reticular fibers. Calcifica- on usually is confined to the matrix. Codman has classified some of these lesions as "benign epiphyseal chondromatous giant-cell tumors" peculiar to the upper end of the humerus.

In the malignant growths, the nuclei of the chondroblasts frequently are of large size, bizarre in shape, and variable in their staining reactions. Mitotic figures are frequent. The calcification is not confined to the matrix but overlaps the cellular elements, unless the section is cut extremely thin. Margins of the tumor may show solid islands of chondroblasts arranged in somewhat alveolar fashion, infiltrating fibrous tissue. Much of the tumor is composed of such alveolar masses with little or no hyaline matrix. Since the division of the chondroblastic tumors into benign and malignant forms on the basis of microscopic study is exceedingly difficult, and since cures have followed curettage plus irradiation in the benign form, it is important that radical operation be postponed until the sections have been reviewed by a competent pathologist. Resection or amputation should not be performed unless the malignant nature of the lesion has been verified. The benign and the malignant forms of chondroblastomas occur in a ratio of 2 to 3. The patients with malignant chondroblastoma usually die within a period of 9 to 18 months. Among the benign lesions followed prior to 1950, all the patients have survived 5 years or more without disease.

CHONDROMATOSIS OF JOINTS

The disease originates in the capsule or the synovial membrane about a joint cavity, and it may also arise in neighboring bursae and tendon sheaths. The present tendency is to regard chondromatosis as a definite neoplastic process, although in the older literature it was confused with free bodies of the joints. Under the microscope are seen various stages of development in which the growth may be traced from mesenchymal cells in the synovial membrane to fetal cartilage, to adult cartilage and thence to osteoid tissue.



FIG. 12. Roentgenogram showing a primary chondrosarcoma involving the upper end of the tibia. Note the translucent character of the periosteal growth and the mottled area in the tibia beneath. In the anterior and lateral views, slight calcification is noted in the soft part tumor.

Chondromatosis of the joints and the bursae is found in adults between 20 and 50 years of age, and it is seen more frequently in males than in females. The joints involved in the order of frequency are: knee, elbow, ankle, hip and shoulder. Involvement of bursae about the knee and the ankle and in the gluteal region has been observed. The symptoms usually are mild but progressive. As a rule, not more than 1 joint is involved.

At operation, glistening pearly bodies, varying in number from a few to more than a hundred, are found free or attached to the synovial membrane.

Roentgenologically, the structures enclosed in the joint capsule show numerous spotted calcareous nodules. At times, in extension of the disease, calcareous stippling may extend along the surface of the bones or for some distance along the neighboring tendons. Occasionally, recurrence may supervene after excision of a synovial membrane or a bursal lining containing these cartilaginous masses. In cases in which there are recurrences, radical surgery is indicated, including amputation, for a few cases have been reported with malignant change.

PRIMARY CHONDROSARCOMA

This large group of malignant tumors contains cartilage in association with a type of myxomatous connective tissue, indicating an origin analogous to benign osteochondromas and chondromas. Primary chondrosarcoma shows its sarcomatous nature from the start. As a rule, it arises periosteally and does not involve the cortex or the medullary cavity of the bone at the onset. Tumors usually occur in patients under 30 years of age; most frequently they are noted in patients from 14 to 21 years of age (the postadolescent period.) The majority of the lesions are situated about the knee in the lower end of the femur or the upper end of the tibia. Distribution of the tumors shows a relationship to points of muscular attachment and to articular regions where cartilage formation persists throughout life. The symptoms usually exist about 5 months before the patient presents himself for examination. Pain is a major complaint; it may or may not follow a relatively mild form of trauma. The pain increases in severity and soon interferes with the function of the part. Stiffness of the joint may soon appear. Weight-bearing becomes painful. Examination reveals swelling in the region of the joint, and a tumor, usually of a rubbery consistency, is palpated. Pathologic fracture is rare. Patients may have fever, leukocytosis and enlargement of significant regional lymph nodes as a late manifestation of the disease.

The roentgenograms show a faintly visible semitranslucent shadow that lifts the periosteum. The wedge-shaped shadow between the cortex and this elevated periosteum is referred to as "lipping" and is an important diagnostic finding. The periosteum is stripped

both at the site of the tumor and below, a few radiating spicules of calcification and slight amounts of new bone being laid down. Involvement of the cortex or the medullary cavity may be extremely slight in early cases, although ultimately this region of the bone is eroded (Fig. 12). This is an unusually malignant form of tumor.

The histopathology of primary chondrosarcoma reveals areas of myxomatous structure changing into early embryonal cartilage with beginning lacunae. In other areas one may see cartilage cells, resembling adult hyaline cartilage with large vesicular nuclei, undergoing mitotic division. Fibrous septa divide the tumor into lobules. Calcification occurs sporadically and irregularly. The myxomatous tissue seen is not a product of cartilaginous degeneration but is an embryonal predecessor of the more mature chondral cells. Myxoma is of great significance in diagnosing the lesion as malignant. Osseous tissue, usually next to the periosteum and overlying the tumor, is either reactive bone or ossification arising in the dense connective tissue strands dipping down from the margins of the tumor. This latter type of ossification usually is sparse in the tumor. Because it is at times extremely difficult to be certain of the presence of chondromyxosarcoma under the microscope, one must be on the alert for the presence of fetal cartilage or myxomatous tissue, the malignant nuclei of the cartilage cells and the occasional presence of cellular areas containing round cells midway between fetal cartilage and chondroblasts (Fig. 13).

Delay in treatment, with prolongation of symptoms prior to operation, diminishes the prospect of a cure. If operative treatment fails to extirpate the disease, primary chondrosarcoma shows a marked tendency to recur locally, as well as to metastasize. Irradiation usually does not affect the progress of the disease, but it may relieve pain when surgery is contraindicated or refused. Amputation, when possible, is the treatment of choice. Radical resection, when used alone, has rarely accomplished a cure.



FIG. 13. Microscopically, the tumor presents areas of myxoma, fetal cartilage and varying degrees of abnormal cartilage cell maturity, associated with mitoses.

TABLE 2. RESULTS OF TREATMENT IN CASES OF PRIMARY CHONDROSARCOMA

Total number of cases	121
Total number of cases followed	114
Number of cases well less than 5 years	11
	103
Total number of fatal cases	88
Number of patients living over 5 years	15
Percentage of 5-year survivals	15%
Number of patients died of disease after 5 years, not included in above 5-year survivals statistics..4 cases:	3.7%

Summary of Treatment
Five-Year Survivals with Primary Chondrosarcoma

Resection	2
Radical excision	1
Amputation	8
Roentgenotherapy and amputation	3
Roentgenotherapy	1
	15 cases

SECONDARY CHONDROSARCOMA

In a consideration of osteochondromas and benign chondromas, it was pointed out that chondrosarcoma might complicate the cartilaginous growths. This secondary malignant change is seen generally in patients of advanced age with osteochondromas and following local recurrence after unsuccessful excision of chondromas in long bones. Most of these tumors occur between 35 and 55 years of age (71 per cent), with the peak of age incidence between 40 and 45 years of age. However, they may occur before 30 years of age. These malignant cartilaginous neoplasms occur most often at the upper end



FIG. 14. Roentgenogram of Paget's disease of the pelvis and the upper femur, showing destructive changes in the greater trochanter and the neck of one of the femurs. This destructive lesion proved to be secondary chondrosarcoma arising in Paget's disease.

of the humerus, about the ribs and at the heel, although they may occur in all other locations noted for *osteochondroma*. The total duration of symptoms averages over 6 years, varying from 2 to 25 years. When sarcomatous change develops secondarily in a benign cartilaginous growth, a history of the earliest phases of the disease usually can be elicited by careful questioning. It may have begun with an injury many years before, the effects of which had subsided. There may be a history of rheumatic pains for many years, or the consciousness that the affected limb always has been crooked or shorter than the corresponding normal member. After an interval of years, and without obvious provocation, pain, swelling or pressure phenomena may appear. It is difficult to establish the actual onset of malignancy in such neoplasms. In some instances, the first lesion obviously is a congenital affair. Judging from the primary form of chondrosarcoma and its subsequent rapid clinical course, the malignant change in benign chondromatous tumors does not precede the onset of the acute symptoms by many months. However, one must be on guard against making a diagnosis of benign osteochondroma on the mere circumstances of the long duration of symptoms in a so-called benign lesion. The degree of pain and the rapidity of tumor growth are more trustworthy guides for diagnosis. The roentgen picture and the biopsy constitute the most conclusive evidence of sarcoma. Pathologic fracture occurs in 6 per cent of secondary chondrosarcomas.

Roentgenologically, the early central secondary chondrosarcomas show evidence of



FIG. 15. Roentgenogram of secondary chondrosarcoma arising from a central chondroma in the ischium, with marked invasion of pelvic soft parts. Note the areas of calcification within the soft part tumor.

the original benign nature of the primary lesion (Fig. 14), with a superimposed malignant change visible as a fuzzy, infiltrating, periosteal shadow. In advanced cases, the entire tumor site becomes the seat of an infiltrating granular mass, with elements of osseous material scattered throughout the process. Stippling in the soft parts as a diagnostic feature has been emphasized by Phemister (see Geschickter & Copeland: Tumors of Bone, for examples). Destruction of cortical bone, with invasion of the medullary cavity, is seen (Fig. 15). It is sometimes extremely difficult to determine roentgenographically the presence of secondary chondrosarcoma developing in a benign exostosis or osteochondroma. In this regard a helpful point is the gradual blotting out of the lines of configuration of the benign lesion from above inward toward the cortical bone. Myositis ossificans and parosteal osteoma may be confusing lesions from the differential diagnostic point of view.

Histopathologically, there are a reduplication and a proliferation of the connective tissue elements. This proliferation takes myxomatous character and becomes spicuous in various parts of the tumor. A from the relatively large amounts of r omatous tissue, secondary chondrosarc differs pathologically in no essential from primary chondrosarcoma. It is c difficult to distinguish between the 2 t of chondrosarcoma in an advanced les Densely cellular and abundant connec tissue strands, interspersed with malig cells near these strands usually borderin myxomatous tissue, and the presence of l malignant multinucleated cartilage cell the tumor lobules are present only in m nant cartilaginous lesions (Fig. 16). secondary chondrosarcoma unsuccess treated, duration of life varies from 2 10 years or more. There may be 1 or n recurrences. Secondary chondrosarc grows more slowly than primary chon sarcoma and is probably influenced by advanced age of the patient.

In many cases of secondary chondro coma it is difficult for the pathologist



FIG. 16. Microscopic section of tumor shown in Figure 15 reveals an area of immature cartilage cells developing into adult tumor cartilage.

render an accurate opinion. The reasons are the histologic gradations that may occur in benign chondromas and, at times, the close resemblance to primary chondrosarcoma. Roentgen studies are of considerable help when interpreting some of these pathologic sections. An estimate can be made of the bone destruction and the extent of the invasion of the marrow cavity by the tumor. The greater the degree of medullary involvement, the worse the prognosis.

Thirty per cent of the adequately treated patients whom we have seen have lived 5 years or more without disease. The majority of the patients successfully treated have had either an amputation, when this was possible, or radical resection. Patients surviving 5 or more years after adequate therapy have been 25 years of age or more. Cures obtained in the upper portion of the humerus or the femur speak for the low grade malignancy of the lesion. Five-year survivals are extremely rare in these locations when involved by primary chondrosarcoma. Only when complete eradication of the tumor cannot be accomplished by radical resection or amputation does irradiation seem to be indicated, and this only as a palliative measure.

TABLE 3. RESULTS OF TREATMENT IN CASES OF SECONDARY CHONDROSARCOMA

Total number of cases	118
Total number of cases followed	107
Number of cases well less than 5 years	21
	86
Total number of fatal cases	60
Number of patients living over 5 years	26
Percentage of 5-year survivals	30%
Number of cases died of disease after 5 years, not included in above 5-year survival statistics ...5 cases:	5.5%

Summary of Treatment

Five-Year Survivals with Secondary Chondrosarcoma

Amputation	12
Excision	3
Resection	4
Resection and irradiation	3
Curettement and irradiation	3
Biopsy and irradiation	1
	26 cases

BIBLIOGRAPHY

Alley, R. G.: Hereditary deforming chondrodysplasia, Radiology 28:544, 1937.

Bennett, G. E., and Berkheimer, G. A.: Malignant degeneration in a case of multiple benign exostoses (with a brief review of the literature), Surgery 10:781, 1941.

Bergstrand, H.: A note on chondroblastic sarcoma, Am. J. Cancer 27:326, 1936.

Campbell, H. E.: Hereditary deforming chondrodysplasia. Report of three cases. J. Bone & Joint Surg. 7:982, 1925.

Carleton, Alice, Elkington, J. St. C., Greenfield, J. G., and Robb-Smith, A. H. T.: Maffucci's syndrome (dyschondroplasia with hemangiomata), Quart. J. Med. 11:203, 1942.

Cleveland, M.: Chondrodysplasia, Surg., Gynec. & Obst. 47:338, 1928.

Codman, E. A.: Epiphyseal chondromatous giant cell tumor of the upper end of the humerus, Surg., Gynec. & Obst. 52:543, 1931.

Cohn, I.: Dyschondroplasia, Ann. Surg. 123:673, 1946.

Coley, B. L.: Neoplasms of Bone, Sec. 2, pp. 90-117, Sec. 3, pp. 231-282, New York, Hoeber, 1949.

Coley, B. L., and Santoro, A. J.: Benign central cartilaginous tumors of bone, Surgery 22:411, 1947.

Coley, W. B., and Coley, B. L.: Primary malignant tumors of the long bones, Arch. Surg. 13:779, 1926, and 14:63, 1927.

Copeland, M. M.: Benign tumors of bone, Surg. Gynec. & Obst. 90:697, 1950.

Copeland, M. M., and Geschickter, C. F.: Bone Tumors and Their Treatment. Monographs on Surgery. Pp. 394-401. New York, Nelson, 1951.

———: Chondroblastic tumors of bone: benign and malignant, Ann. Surg. 129:724, 1949.

Creyssel, J., and Peycelon, R.: Osteogenic multiple exostosis terminated by malignant development of chondroma, Lyon chir. 27:733, 1930.

Dahl, B.: Relation between chondrodysplasia, multiple chondromatosis and Ollier's disease, Acta orthop. scandinav. 1:127, 1930.

Ehrenfried, A.: Hereditary deforming chondroplasia. Multiple cartilaginous exostoses: a review of the American literature and report of twelve cases. J.A.M.A. 68:502, 1917.

Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, ed. 3, Part 2, pp. 37-180, Philadelphia, Lippincott, 1949.

Hammarström, S: Ein Fall von chondroblastischem Sarkom, Acta radiol. 15:668, 1934.

Huber, J., and Belot, D.: Chondromatosis with multiple and unilateral localizations, Bull. et mém. Soc. méd. hôp Paris 54:388, 1930.

Jacobson, S. A.: *Contribution to the pathogenesis* of multiple hereditary osteochondromatosis, Am J. Cancer 39:220, 1940.

Jaffe, H. L.: Hereditary multiple exostoses, Arch. Path. 36:335, 1943

Jaffe, H. L., and Lichtenstein, L: Benign chondroblastoma of bone, Am. J. Path. 18: 969, 1942

Jones, H. T.: Loose body formation in synovial osteochondromatosis, with special reference to etiology and pathology, J. Bone & Joint Surg. 6:407, 458, 1924.

Kartal, S: Chondromatosis of joint capsule, Surg, Gynec. & Obst. 51:99, 1930

Kolodny, A.: Bone sarcoma, Surg, Gynec & Obst 44:1-214, 1927.

Kuzma, J. F., and King, J. M: Dyschondroplasia with hemangiomatosis (Maffucci's syndrome), Arch. Path. 46:74, 1949.

Langenskiold, A.: Normal and pathologic bone growth in the light of the development of cartilaginous foci in chondrodysplasia, Acta chir scandinav. 95.367, 1947.

Léri, A., and Weil, J.: Cinq cas d'osteochondromatose articulare, Bull et mém. Soc. méd. hôp. Paris 46:1492, 1930.

Leriche, R., and Brenckmann, E.. Pathogenesis of chondromatosis, dead bone in humeral epiphyses of elbow in case of chondromatosis, Lyon chir. 26:844, 1929.

Lichtenstein, L, and Jaffe, H L: Chondrosarcoma of bone, Am J. Path. 19:553, 1943.

Mahorner, H. R Dyschondroplasia, J. Pediat. 10:1, 1937.

Morton, J J., and Mider, C. B Chondrosarcoma, Ann Surg. 126:895, 1947.

Moutier, G.: Chondro-sarcome de l'humérus avec ostéite éburnante, Bull et mém Soc. nat. de chir 56:1205, 1930

Phemister, D B Chondrosarcoma of bone, Surg., Gynec & Obst 50:216, 1930.

———: Conservative surgery in the treatment of bone tumors, Surg., Gynec & Obst. 70: 355, 1940.

Putti, Vittorio: Malignant bone tumors, Surg., Gynec. & Obst. 48:324, 1929.

Stein, I., Stein, R O., and Beller, M. L.: Living Bone in Health and Disease, pp. 117-131, Philadelphia, Lippincott, 1955.

Stocks, P., and Barrington, A.: Hereditary Disorders of Bone Development. In the Treasury of Human Inheritance. Edited by K. Pearson. 22 vol. Vol. 3, 182 pp. London, Cambridge, 1925.

Wolf, J.: Chondromata of para-articular bursae, Ztschr. orthop. Chir. 52:629, 1930.

Tumores de Origine Cartilaginose

Summario in Interlingua

Le presente reporto include un discussion del processos embryonic que es involvite in le formation de osso. Es presentate un classification del entitates morbose. Le entitates morbose es discutite in detalio insimul con *le tractamento melio adaptate pro* cata forma de processo de tumor cartilaginose.

Es signalate plure aspectos, importante ab le puncto de vista del formation de tumores, tanto del embryologia como etiam del subsequente crescentia de precartilaginose histos conjunctive. Le majoritate del benigne e maligne tumores cartilaginose de osso occurre durante le adolescentia e se trova in le triangulo justo in retro del platta cartilaginose ubi le crescentia osse es effectuate.

Cartilaginose tumores osse es relationate a certe phases del disveloppamento skeletal. Le crescentia cartilaginose es representate per le chondromas, le chondromyxomas, le chondromatoses de synovio articulari, e le chondrosarcomas. Combinationes de crescentia cartilaginose, vascularisation (con resorption per vasos e cellulas gigante de *cartilagine calcificate in le production* final del osso), e ossification de histos fibrose es representate per chondroblastoma e le osteochondromas. Chondromatosis de articulationes e chondroma es etiam relationate a un plus primitive phase cartilaginose.

3

Osteogenic Sarcoma of Bone

HENRY L. JAFFE, M.D.*

Osteogenic sarcoma of bone is a specialized connective tissue sarcoma that starts in the interior of the affected bone and is distinguished by the formation of neoplastic osteoid and osseous tissue. This tissue is formed out of the sarcomatous connective tissue directly and (in some tumors or tumor areas) also by way of an intermediary cartilaginous stage. The amount of neoplastic osteoid and osseous tissue formed in an individual osteogenic sarcoma may be meager, but it is more likely to be considerable, and it may even be very great.

On the basis of this definition, a malignant connective tissue sarcoma of bone that does not give evidence of osteogenesis (either at its original site or in any metastases) is to be interpreted as a fibrosarcoma of bone rather than an osteogenic sarcoma of bone. Osteogenic sarcoma also is to be distinguished from chondrosarcoma of bone, since a chondrosarcoma develops out of cartilage directly and remains essentially a malignant tumor of cartilage. The conventional osteogenic sarcoma also is to be differentiated from the lesion we call *juxtacortical osteogenic sarcoma*—a distinctive bone-forming tumor that develops in relation to the periosteum and/or immediate parosteal connective tissue.

As to nomenclature, *osteosarcoma* often is used synonymously with, or even in preference to, *osteogenic sarcoma*. The tendency to qualify the name of a particular osteogenic sarcoma by such prefixes as fibro-, chondro-, and/or myxo- long has been on the wane. The tendency persists to subdivide the osteogenic sarcomas into medullary and subperiosteal, telangiectatic, sclerosing, periosteal, etc., but it does not seem to be of much utility either for understanding the basic character of osteogenic sarcoma or for evaluating the probable course in a given case.

CLINICAL FEATURES

Osteogenic sarcoma is the most common of the primary malignant tumors of bone. As to age distribution, about three fourths of the patients in any representative series are between 10 and 25 years, some below 10, though few below 5, and most of the rest between 26 and 40 years. As to sex, males seem to preponderate over females in the proportion of approximately 2 to 1.

It is the long bones of the limbs that are the most common sites of osteogenic sarcoma, the lesion being in a femur in more than half of all cases, and in a tibia or a humerus in many of the others. All other localizations taken together account for only a small minority of the cases in any large series. When a femur is involved, the lesion is more likely to be in the lower part of the bone, and, when a tibia or a humerus is involved, in the upper part of the bone.

The cardinal, and nearly always the initial, complaint in cases of osteogenic sarcoma is local pain. This usually is reported as grad-

* From the Laboratory Division of the Hospital for Joint Diseases, New York 35, N. Y.

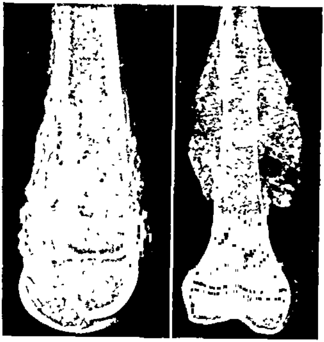


FIG. 1. (*Left*) A densely ossified osteogenic sarcoma in its characteristic location at the end of a long bone shaft At a level deep to the cut surface shown, the tumor had penetrated the epiphysial cartilage plate and extended into the epiphysis (*Right*) A moderately ossified osteogenic sarcoma that is situated farther along the shaft and does not reach the epiphysial plate at all.

ually increasing and becoming more persistent over a period of a few months, and as being associated with the development of a local swelling. If the tumor has extended to a joint capsule, there is likely to be some limitation of motion in the affected area, and fluid may be present in the joint. If the tumor is in a bone of a lower limb, there often also is complaint of a limp. Trauma, though often mentioned in connection with the lesion, seems to bear no causal relation to its occurrence.

A patient having an osteogenic sarcoma often shows a somewhat elevated serum alkaline phosphatase value. Ablation of the affected limb usually is followed promptly by a drop in the alkaline phosphatase, though often not to completely normal values. A subsequent sharp rise in the alkaline phosphatase value is an indication that, despite ablation, the tumor has metastasized. The alkaline phosphatase value may rise even before the metastases are visible roentgenographically in the lungs. After the development of metastases, the duration of life usually is only a matter of a few months.

At best, osteogenic sarcoma is a tumor having an extremely high mortality rate—about 90 to 95 per cent. Prognostic significance sometimes has been attached to the intensity of ossification shown by the primary tumor. Actually, however, the disease usually reaches a fatal termination about as quickly when this tumor is a highly ossified one as when it is not. However, in contrast with ossification, location does have some importance in the prognosis. In particular, so far as location in limb bones is concerned, the prognosis is somewhat less grave when the lesion is distal than when it is close to the trunk of the body

ROENTGENOGRAPHIC AND GROSS PATHOLOGIC FEATURES

Though starting in the interior of the affected bone, most osteogenic sarcomas, when first seen, not only involve the interior but somewhere have already penetrated the cortex to lie beneath the periosteum; or they may even have perforated the latter in some places and gone on growing beyond it. The gross pathology of an osteogenic sarcoma intact in its setting is described best as it appears in long bones of limbs. Usually, the lesion involves one end of the shaft and at least part of the adjacent epiphysial area, though sometimes it is situated well along toward the middle of the shaft (Fig. 1). At the end directed toward the middle of the shaft, generally the tumor terminates in a more or less dome-shaped plug in the medullary cavity. At the opposite end—that is, in the direction of the joint—the delimitation of the tumor is far less precise. It often reaches the epiphysial end of the bone, and,

to a lesser or a greater degree, it may even extend to beneath the articular cartilage.

As already noted, an individual osteogenic sarcoma may show relatively little, moderate or very considerable ossification. In our series of cases, about one quarter of the tumors were relatively slightly ossified, about one quarter moderately ossified, and one half heavily ossified and considerably eburnated. The degree of ossification seems to have little to do with the size or the stage of development of the lesion. That is, a tumor which is still small and confined mainly within the limits of the bone may be slightly or highly ossified, and a tumor which is large and has erupted widely beyond the bone limits may likewise be so (Fig. 2).

In a small confined lesion showing relatively little ossification, the tumor tissue tends to be rather firm and elastic and whitish on the whole, but also to show gritty

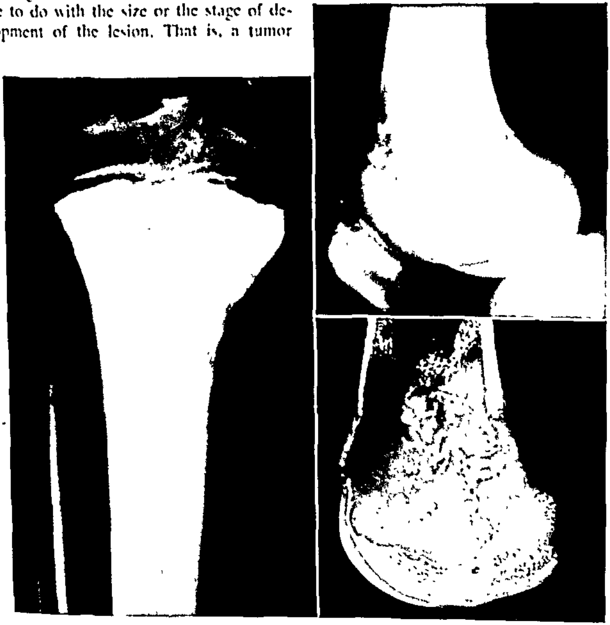


FIG. 2. (*Left*) Roentgenograph of a highly ossified osteogenic sarcoma that still is confined largely within the bone, though the cortex on the medial side is being destroyed because of some penetration of the tumor outward from the bone. (*Right, top*) Roentgenograph of an osteogenic sarcoma that is producing only little tumor osteoid and tumor bone. (*Right, bottom*) Roentgenograph of a thin slice of the lesion shown above. This slice demonstrates far better than the clinical roentgenograph the ossification taking place within the tumor.

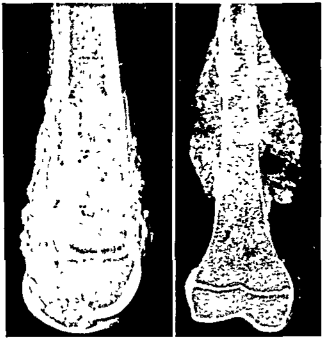


FIG 1. (*Left*) A densely ossified osteogenic sarcoma in its characteristic location at the end of a long bone shaft. At a level deep to the cut surface shown, the tumor had penetrated the epiphysial cartilage plate and extended into the epiphysis. (*Right*) A moderately ossified osteogenic sarcoma that is situated farther along the shaft and does not reach the epiphysial plate at all.

ually increasing and becoming more persistent over a period of a few months, and as being associated with the development of a local swelling. If the tumor has extended to a joint capsule, there is likely to be some limitation of motion in the affected area, and fluid may be present in the joint. If the tumor is in a bone of a lower limb, there often also is complaint of a limp. Trauma, though often mentioned in connection with the lesion, seems to bear no causal relation to its occurrence.

A patient having an osteogenic sarcoma often shows a somewhat elevated serum alkaline phosphatase value. Ablation of the affected limb usually is followed promptly by a drop in the alkaline phosphatase, though often not to completely normal values. A subsequent sharp rise in the alkaline phosphatase value is an indication that, despite ablation, the tumor has metastasized. The alkaline phosphatase value may rise even before the metastases are visible roentgenographically in the lungs. After the development of metastases, the duration of life usually is only a matter of a few months.

At best, osteogenic sarcoma is a tumor having an extremely high mortality rate—about 90 to 95 per cent. Prognostic significance sometimes has been attached to the intensity of ossification shown by the primary tumor. Actually, however, the disease usually reaches a fatal termination about as quickly when this tumor is a highly ossified one as when it is not. However, in contrast with ossification, location does have some importance in the prognosis. In particular, so far as location in limb bones is concerned, the prognosis is somewhat less grave when the lesion is distal than when it is close to the trunk of the body.

ROENTGENOGRAPHIC AND GROSS PATHOLOGIC FEATURES

Though starting in the interior of the affected bone, most osteogenic sarcomas, when first seen, not only involve the interior but somewhere have already penetrated the cortex to lie beneath the periosteum; or they may even have perforated the latter in some places and gone on growing beyond it. The gross pathology of an osteogenic sarcoma intact in its setting is described best as it appears in long bones of limbs. Usually, the lesion involves one end of the shaft and at least part of the adjacent epiphysial area, though sometimes it is situated well along toward the middle of the shaft (Fig. 1). At the end directed toward the middle of the shaft, generally the tumor terminates in a more or less dome-shaped plug in the medullary cavity. At the opposite end—that is, in the direction of the joint—the delimitation of the tumor is far less precise. It often reaches the epiphysial end of the bone, and,

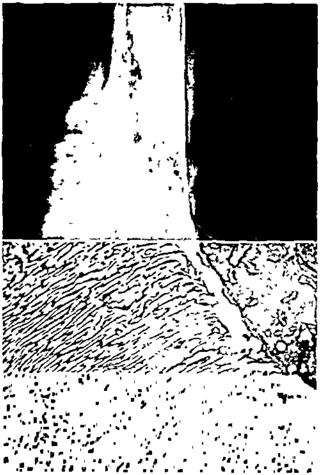


FIG. 3. (*Top*) Roentgenograph of a slice of a femur that was the site of an osteogenic sarcoma. Note the transverse ("sunburst") striations in the tumor tissue beyond the cortex. (*Bottom*) Photomicrograph showing, under very low power, the architecture of the tumor bone that courses transversely through the extra-osseous tumor mass to produce the shadow visible in the roentgenograph above. A small fragment of the femoral cortex is to be seen in the upper right-hand corner of the picture

yellowish dots and streaks where it is undergoing ossification. In a large erupted and still relatively unossified lesion, the tumor tissue is likely to be extensively necrotic, cystified and telangiectatic. Furthermore, one finds that, in these latter lesions, the cortex has undergone extensive destructive resorption and perhaps been so weakened that a pathologic fracture had resulted

In a small confined and highly ossified lesion, the tumor area is dense, compact and eburnated practically throughout. In a large and highly ossified tumor, on the other hand, the most densely ossified portion will be found in the interior of the affected bone area, while the tissue overlying the cortex, though quite ossified on the whole, will show at least some parts, especially near the periphery, which as yet are ossified only slightly, if at all. In such a lesion, the highly ossified tumor areas tend to have a yellow-white color and the hardness of cortical bone; the less ossified areas tend to be less yellow and more white and to have a rather gritty-rubbery consistency; and the least ossified areas tend to be white and of a chondroid or a fleshy consistency.

Very frequently, one can make a preoperative diagnosis of osteogenic sarcoma from the roentgenographic findings. The greater the degree of ossification of the lesion, the easier it is to do this, and it is only the very meagerly ossified lesions that are likely to be completely baffling. In the cases in which there is considerable osteogenesis in the tumor, the shadow cast by the affected bone area and by any tumor tissue beyond the limits of the bone proper is densely radiopaque, at least to a very large extent. When there is considerable ossified tumor tissue beyond the limits of the cortex, one may note transverse or radiating striations extending out from the cortex in a "sunburst" pattern. As histologic examination reveals, such striations represent osseous trabeculae coursing transversely through less heavily calcified and ossified extracortical tumor tissue (Fig. 3).

Spread of the tumor from the original bone site to the regional lymph nodes seems to be of rare occurrence. Distant metastasis seems to occur mainly, if not exclusively, by the hematogenous route. Metastases to the lungs are found consistently in cases which have terminated fatally, and often they are the only metastatic finding. However, occasionally one or more skeletal metastases are observed along with them (and perhaps with



FIG 5 (*Top*) Photomicrograph (× 125) showing the emergence of tumor bone in the sarcomatous stroma Note that the sarcomatous stromal cells are becoming enmeshed in the ground substance and developing into the osteocytes of the tumor bone. (*Bottom*) Photomicrograph (× 125). Note the development of tumor osteoid and tumor bone in the sarcomatous stroma and the attachment of the ossified tumor tissue to a trabeculum of the original bone.

shaped, or they may present a highly variegated anaplastic morphology, or they may be roundish and lie in lacunae so that the appearance of the regional sarcomatous stroma suggests cartilage. In the anaplastic areas, most of the cells contain two or more large and sometimes bizarre hyperchromatic nuclei, and the large cells particularly may even be crowded with such nuclei, appearing as veritable sarcoma giant cells. In the cartilaginous areas one can note transition forms that indicate that the tumor cartilage has been created directly through differentiation of the sarcomatous spindle cells (Fig. 4).

Where the sarcomatous stromal tissue is beginning to undergo ossification, one notes the appearance of intercellular fibrillar collagenous material, at first mainly between small groups of the stromal cells. Where there has been further advance toward ossification, one notes increase of the intercellular substance in the form of streamers that tend to break up the cell clusters so that individual cells become more widely separated. Many of the sarcomatous stromal cells become enmeshed in the ground substance, and one then sees sheets and trabeculae of tumor osteoid Further along the line of evolution, the osteoid trabeculae and the sheets of osteoid show deposits of calcium in the intercellular matrix, inaugurating the appearance of actual tumor bone (*Fig 5*) *It is of interest to note that*, after becoming incarcerated in the trabeculae of tumor osteoid and tumor bone, the originally plump sarcomatous stromal cells tend to become smaller. Indeed, as Phemister already has pointed out, the more ossified the tumor bone trabeculae become, the smaller and the more sparse, on the whole, are the tumor cells contained within them, and the more closely do these cells approach in appearance the osteocytes of nontumor bone. That is, this process of incarceration tends to have a *normalizing* influence, as it were, upon the appearance of the tumor cells that have become the osteocytes of the tumor bone.

If the lesion is one whose sarcomatous stroma does not lay down much tumor osteoid and bone, the original osseous tissue at the site of its growth is subjected from the beginning mainly to resorption and dissolution. Specifically, as the tumor tissue fills the marrow spaces and surrounds the original spongy trabeculae, these tend to be gnawed out and broken up irregularly, and many are resorbed altogether, while at the same time the cortex, too, is resorbed and



FIG. 5. (*Top*) Photomicrograph (× 125) showing the emergence of tumor bone in the sarcomatous stroma Note that the sarcomatous stromal cells are becoming enmeshed in the ground substance and developing into the osteocytes of the tumor bone. (*Bottom*) Photomicrograph (× 125). Note the development of tumor osteoid and tumor bone in the sarcomatous stroma and the attachment of the ossified tumor tissue to a trabeculum of the original bone.

shaped, or they may present a highly variegated anaplastic morphology, or they may be roundish and lie in lacunae so that the appearance of the regional sarcomatous stroma suggests cartilage. In the anaplastic areas, most of the cells contain two or more large and sometimes bizarre hyperchromatic nuclei, and the large cells particularly may even be crowded with such nuclei, appearing as veritable sarcoma giant cells. In the cartilaginous areas one can note transition forms that indicate that the tumor cartilage has been created directly through differentiation of the sarcomatous spindle cells (Fig 4).

Where the sarcomatous stromal tissue is beginning to undergo ossification, one notes the appearance of intercellular fibrillar collagenous material, at first mainly between small groups of the stromal cells. Where there has been further advance toward ossification, one notes increase of the intercellular substance in the form of streamers that tend to break up the cell clusters so that individual cells become more widely separated. Many of the sarcomatous stromal cells become enmeshed in the ground substance, and one then sees sheets and trabeculae of tumor osteoid. Further along the line of evolution, the osteoid trabeculae and the sheets of osteoid show deposits of calcium in the intercellular matrix, inaugurating the appearance of actual tumor bone (Fig 5) It is of interest to note that, after becoming incarcerated in the trabeculae of tumor osteoid and tumor bone, the originally plump sarcomatous stromal cells tend to become smaller. Indeed, as Phemister already has pointed out, the more ossified the tumor bone trabeculae become, the smaller and the more sparse, on the whole, are the tumor cells contained within them, and the more closely do these cells approach in appearance the osteocytes of nontumor bone. That is, this process of incarceration tends to have a *normalizing* influence, as it were, upon the appearance of the tumor cells that have become the osteocytes of the tumor bone.

If the lesion is one whose sarcomatous stroma does not lay down much tumor osteoid and bone, the original osseous tissue at the site of its growth is subjected from the beginning mainly to resorption and dissolution. Specifically, as the tumor tissue fills the marrow spaces and surrounds the original spongy trabeculae, these tend to be gnawed out and broken up irregularly, and many are resorbed altogether, while at the same time the cortex, too, is resorbed and

destroyed substantially. On the other hand, if the lesion is one in which the deposition of neoplastic osseous tissue is considerable, the intertrabecular marrow spaces come to contain much tumor bone, and the original spongy trabeculae become encased by it, yielding slowly to it by a process of gradual substitution. In such a case, the process of



FIG 6 (*Top*) Photomicrograph (× 60) showing the formation of considerable tumor bone in intertrabecular marrow spaces (*Bottom*) Photomicrograph (× 60) showing even more considerable tumor-bone formation. It is understandable why lesions presenting such extensive tumor-bone formation are intensely radiopaque in the roentgenograph.

erosion and destruction of the original cortex, too, is slower than in a case in which the osteogenic potentiality of the sarcoma is relatively slight (Fig. 6).

In an individual case, before the tumor tissue has yet extended through the cortex, the lesional area already may show some new bone laid down under the periosteum. Such a deposit, which represents merely a reaction to periosteal irritation, varies in thickness. It consists of loosely woven radiating bone trabeculae interspersed with moderately vascular loose-meshed connective tissue apparently quite free of tumor cells. After the sarcomatous stromal tissue has spread beyond the limits of the cortex, the bone found under the periosteum represents mainly tumor bone. In an individual case, one may even find a considerable amount of tumor bone under the periosteum, distending it, sometimes tremendously, and even penetrating it, perhaps in many places. It is when the trabeculae in this subperiosteal tumor tissue run at right angles to the longitudinal axis of the bone that the roentgenogram presents the "sunburst" pattern already mentioned (see Fig. 3).

Finally, it should be noted that most osteogenic sarcomas are rather vascular. They are more so in some portions than in others, the still-cellular stromal tissue being interspersed with smaller or larger sinuous thin-walled blood channels bordered by tumor cells In some parts in particular, one may note engorged vascular channels lying close together Viable tumor cells usually border them, and some tumor cells also may be found admixed with the blood in the spaces. Such telangiectatic areas may be near, or independent of, areas in which the tumor tissue is undergoing anemic or even hemorrhagic necrosis.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

As already implied the diagnosis of osteogenic sarcoma is often not difficult Specifically, one is almost certainly dealing with an osteogenic sarcoma if the lesion is a substantially radiopaque tumor that has broken out, or is breaking out, from the interior of the shaft end of a long limb bone and the patient is between 10 and 25 years of age If the patient is within these age limits, and if the conditions relating to location and manner of advance of the lesion also are fulfilled, the tumor is still likely to be an osteogenic sarcoma, even if the lesional area is only spottily radiopaque. The problems of differential diagnosis are mainly the problems of distinguishing between: (1) juxtacortical osteogenic sarcoma and conventional osteogenic sarcoma; (2) essentially non-bone-forming sarcomas (for instance, fibrosarcoma) and osteogenic sarcomas forming relatively little tumor osteoid and bone; (3) chondrosarcoma and osteogenic sarcoma; and (4) certain benign lesions that may show radiopacity in the lesional tissue (for instance, a highly ossified focus of fibrous dysplasia) and osteogenic sarcoma

JUXTACORTICAL OSTEOGENIC SARCOMA

As already indicated, the juxtacortical osteogenic sarcoma is a distinctive bone-forming tumor that develops in relation to the periosteum and/or immediate parosteal connective tissues. As the term *juxtacortical* implies, it is a tumor which, at least at first, is merely oriented to the regional bone, in the sense of starting just beyond the confines of the cortex. In this respect, the lesion is to be differentiated from conventional osteogenic sarcoma of bone, which originates within the affected bone. From the clinical point of view also, it is worth while to distinguish between them, because of the much better over-all prognosis of juxtacortical osteogenic sarcoma.

Juxtacortical osteogenic sarcoma is a much less common lesion than conventional osteogenic sarcoma. It seems to show some predilection for females. The age range of the patients is fairly wide, and older adults are fairly well represented among them. In fully half the cases, the lesion is in the lower femoral area In other cases, the site usually

is in relation to some other tubular bone—generally likewise toward the end rather than in the mid-shaft area.

The earlier stages of the lesion are represented roentgenographically by the presence of a more or less radiopaque shadow in the soft tissues overlying the cortex of the neighboring bone. The parosteal mass varies in

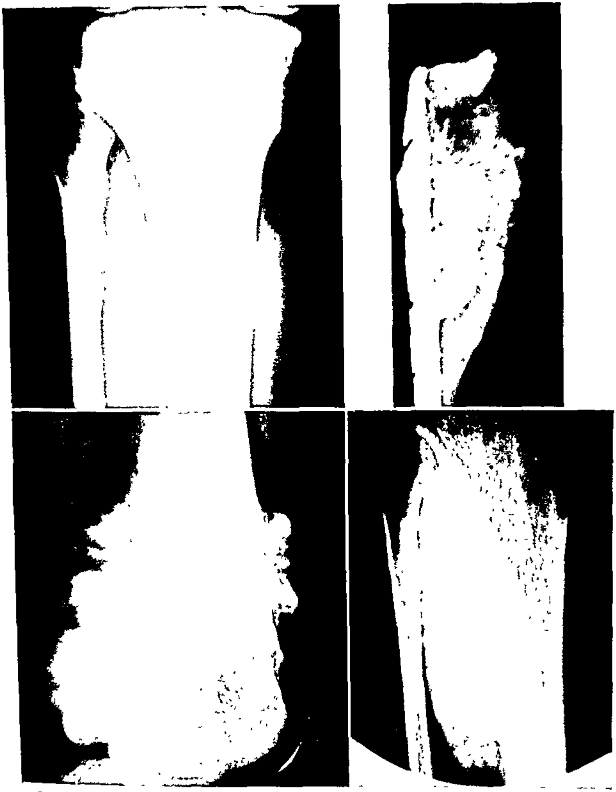


FIG 7. (*Top, left*) Roentgenograph of a juxtacortical osteogenic sarcoma abutting on the medial surface of a tibia. (*Top, right*) Roentgenograph of a slice of the resection specimen in this case (*Bottom, left*) Roentgenograph of a juxtacortical osteogenic sarcoma. The picture shows the third recurrence within a period of 4 years. (*Bottom, right*) Roentgenograph of a juxtacortical osteogenic sarcoma. This was the appearance of the lesion when the patient presented herself for treatment.

drosarcoma has developed *de novo* or from a pre-existing cartilage growth, the tumor, if left to run its natural course, ultimately will break out of the confines of the cortex, and a definite mass will be visible roentgenographically beyond the limits of the altered cortex. If the affected bone is sectioned in its long axis, it will be found, whether the tumor is bulky or not, that the pathologic tissue in the interior of the bone, and any which is outside the limits of the cortex, is composed of smaller or larger faceted islands of cartilage. If radiopacities are visible roentgenographically in the lesional area, these will turn out to have represented areas in which the tumor cartilage is heavily calcified or even ossified. A rather bulky chondrosarcoma is likely also to present evidences of secondary degeneration. Specifically, one may note smaller and larger areas in which the cartilaginous tumor tissue is soft and mucoid, or even frankly cystified. Also, one may note areas in which the tumor tissue has undergone hemorrhagic necrosis, and even softening on that account.

As to the microscopic findings in this connection, one must bear in mind that the basic difference between the 2 lesions is that chondrosarcoma issues out of cartilage, while osteogenic sarcoma issues from more primitive tissue, and specifically from sarcomatous osteogenic connective tissue. Nowhere does a chondrosarcoma show, cytologically, as do all osteogenic sarcomas properly so called, neoplastic osteoid and osseous tissue evolving directly from a sarcomatous connective tissue stroma.

FIBROUS DYSPLASIA

It is only in relation to a solitary focus of fibrous dysplasia that any danger of confusion with osteogenic sarcoma may arise It is more likely to arise in regard to those solitary fibrodysplastic lesions in which the replacement tissue in the lesional area is rather on the fibrous side of the fibro-osseous complex than on the osseous side. Whether the replacement tissue tends toward the osseous or toward the fibrous side, lesions of fibrous dysplasia do not tend to perforate the cortex, though often they thin the cortex and expand the contour of the affected bone part. Furthermore, the fibrous stroma shows no evidences of atypism or remarkable plumpness of the cells such as would indicate that the stroma is sarcomatous. Nevertheless, there can be no doubt that, in an occasional instance, a solitary focus of fibrous dysplasia is misinterpreted as an osteogenic sarcoma.

TREATMENT

In the management of osteogenic sarcoma, amputation appears to be the procedure of choice for lesions that are accessible surgically (as the great majority are, since they occur in long bones of limbs). It is generally, though not universally, held that the amputation should be done as soon as possible *after the diagnosis has been established unequivocally* through examination of tissue obtained by needle punch or incision. Apparently, in most cases, nothing is to be gained by a preliminary course of local radiation therapy or by prophylactic radiation of the chest after amputation

As to the proper level for the amputation, one usually can clear the tumor if the amputation is done 4 or 5 inches above the roentgenographically established upper limit of the lesion. However, one is on much safer ground if one carries out the amputation even higher up. For example, the author has seen several instances of osteogenic sarcoma of the lower end of the femur in which, had not a disarticulation at the hip been done, there would certainly have been a local recurrence in the stump. This was shown by the fact that examination of the ablated limb revealed a focus of tumor tissue in the upper end of the femur, separated from the tumor tissue in the lower end by a large area of apparently uninvolved femoral shaft. For an osteogenic sarcoma in the upper end of a femur, the procedure should be more radical than a disarticulation at the hip joint, per-

haps even a hemipelvectomy, though the prognosis is gloomy at best when the tumor is at this site. For an osteogenic sarcoma of the humerus, an interscapulothoracic amputation is indicated, but again, in accordance with the location near the trunk, the outlook is extremely poor.

Several cases have been reported in which a limb was ablated because of an osteogenic sarcoma, in which a focus of pulmonary metastasis subsequently appeared, and in which pneumonectomy was followed by a survival period of 5 years or more. The reasonable explanation for such cases is that the pulmonary metastasis in question was still the only one, and that it was removed before there could be retrograde pulmonary seeding from it.

BIBLIOGRAPHY

Case Records of the Massachusetts General Hospital: Osteogenic sarcoma of humerus, with ossifying metastases in regional nodes, New England J. Med. 225:953, 1941.

Ewing, J.: Review of classification of bone tumors, Surg., Gynec. & Obst. 68:971, 1939.

Ferguson, A. B.: Treatment of osteogenic sarcoma, J. Bone & Joint Surg. 22:92 and 916, 1940.

Goldenberg, R.: Eight-year survival of osteogenic sarcoma of the tibia, with pulmonary metastasis, Bull. Hosp. Joint Dis. 15:67, 1954.

Hodges, P. C., Phemister, D. B., and Brunschwig, A.: The Roentgen-Ray Diagnosis of Diseases of the Bones and Joints, p. 181, New York, Nelson, 1938.

Jaffe, H. L.: Tumors of the skeletal system: pathologic aspects, Bull. New York Acad. Med. 23:497, 1947.

Jaffe, H. L., and Bodansky, A.: Diagnostic significance of serum alkaline and acid phosphatase values in relation to bone disease, Bull. New York Acad. Med. 19:831, 1943.

Jaffe, H. L., and Selin, G.. Tumors of bones and joints, Bull. New York Acad Med. 27: 165, 1951.

Luck, J. V.: A correlation of roentgenogram and pathologic changes in ossifying and chondrifying primary osteogenic neoplasms, Radiology 40:253, 1943.

Pendergrass, E. P., Lafferty, J. O., and Horn, R. C.: Osteogenic sarcoma and chondrosarcoma, with special reference to roentgen diagnosis, Am. J. Roentgenol. 51:234, 1945.

Phemister, D. B.: A study of the ossification in bone sarcoma, Radiology 7:17, 1926.

Price, C. H. G.: The grading of osteogenic sarcoma, Brit. J. Cancer 6:46, 1952.

Woodard, H. Q., and Higinbotham, N. L.: The correlation between serum phosphatase and roentgenographic type in bone disease, Am. J. Cancer 31:221, 1937.

Sarcoma Osteogene de Osso

Summario in Interlingua

Sarcoma osteogene de osso es un specialisate sarcoma de histos conjunctive que comencia al interior del osso afficite e se distingue pathologicamente per le formation de neoplastic histos osteoide e ossee. Iste histos es formate ab le sarcomatose histos conjunctive directemente e etiam (in certe tumores o areas tumoral) via un intermediari stadio cartilaginose. A vices le quantitate del neoplastic histos osteoide e ossee que es formate in le sarcoma osteogene individual pote esser magre, sed plus frequentemente illo es considerabile; illo etiam pote esser grande.

Le diagnose clinic de sarcoma osteogene es frequentemente facile. Specificamente, on pote esser quasi certe de esser in le presentia de un sarcoma osteogene si le lesion in question es un tumor de plus o minus complete radio-opacitate que erumpe o ha erumpite ab le interior del extremitate diaphysic de un osso longe e si le patiente ha un etate de inter 10 e 25 annos. Si le etate del patiente es intra le limites mentionate e si etiam le conditiones de location e de forma de progredimento del lesion es complite, le tumor es probabilemente un sarcoma osteogene, mesmo si le area del lesion exhibi solmente un radio-opacitate sporadic.

Le problemas del diagnose differential es principalmente le problemas del distinction (1) inter sarcoma osteogene juxtacortical e sarcoma osteogene conventional, (2) inter sarcomas intrinsecamento non osteoplastic

(per exemplo fibrosarcoma) e sarcomas osteogene que forma relativemente pauc histos tumoral osteoide e ossee, (3) inter chondrosarcoma e sarcoma osteogene, e (4) inter certe lesiones benigne que exhibi radioopacitate in le histos lesional (per exemplo un altemente ossificate foco de dysplasia fibrose) e sarcoma osteogene.

4

Osteogenic Sarcoma of Bone

IRVIN STEIN,* M.D., F.A.C.S., F.I.C.S., AND MARTIN L. BELLER,* M.D.

Osteogenic sarcoma of bone is the most frequent primary malignant tumor of bone. It arises from proliferating, often anaplastic, primitive bone-forming mesenchyme. It must be differentiated sharply from chondrosarcoma, which arises from mature cartilage, and from fibrosarcoma, which has no bone-forming elements.

INCIDENCE

Osteogenic sarcoma is more common in males than in females, and it is found most frequently between the ages of 10 and 25. However, it may occur at any age. Most often when it is found after the age of 40, it is malignant supervention in Paget's disease.[8] Statistical incidence is not as small as many believe. It is described as occurring 1 in each 107,000 of the population.[2]

LOCATION

This tumor is found most commonly in the distal femur, the proximal femur and the proximal humerus. It is observed more rarely in other long bones, and even in the flat bones, especially the pelvis.

ETIOLOGY

The cause of this, as of other primary tumors, is unknown. However, it is of interest that numerous cases have been reported following radiation—by roentgenotherapy, radium irradiation[6] or injection of radium.[9]

The predisposition for this tumor in people with Paget's disease also is known.[10]

It has been described as occurring in 2 cases following fracture.[2] When one considers a fresh fracture occurring in a patient with an osteogenic sarcoma in an early stage,[7] the more reasonable conclusion is that it is a pure coincidence or perhaps a pathologic fracture occurring in bone weakened by this tumor.

CLINICAL FEATURES

Pain eventually becomes a prominent symptom. Unfortunately, early in the process there may be no pain or, at most, a vague ache.

Swelling is a late sign and indicates extension beyond the anatomic confines of the involved bone. At this stage dilation of veins, increased heat and even pathologic fracture may be found.

Systemic symptoms may include fever, malaise and progressive debilitation.

Metastatic changes on the basis of venous metastasis may present with masses in the groin or the axilla or with signs of pulmonary involvement. Unfortunately, these metastatic foci may already be well established by the time the tumor is diagnosed.

ROENTGENOGRAPHIC FEATURES

The major features are those of bone destruction, bone formation and periosteal proliferative reaction.

The salient feature is bone destruction. Superimposed upon this is new tumor-bone formation within the bone itself. The periosteal reaction may occur simply as a result

* Philadelphia, Pa.



FIG. 1. Osteogenic sarcoma. Adolescent subject. Classic osteoblastic lesion characterized by condensation in the distal femoral metaphysis It does not appear to cross the epiphysial line. At the flare of the shaft an area of decreased density suggesting bone destruction is observed. The medial aspect of the lower shaft shows considerable periosteal lifting with new bone formation. This suggests invasion of the subperiosteal area by tumor cells extending through the haversian canals (Dr. M M. Pomeranz)

of the lifting of the periosteum by the underlying tumor growth, or it may represent actual tumor-bone formation in the subperiosteal region (Fig. 1).

These characteristics have led to the following subdivisions as listed in the 1939 classification of the Bone Sarcoma Registry of the American College of Surgeons:

1. Medullary and subperiosteal
2. Telangiectatic
3. Sclerosing
4. Periosteal

The tumor that forms little new bone is termed the osteolytic variant of osteogenic sarcoma. Conversely, the tumor that forms much new bone is termed the osteoblastic or sclerosing variant of osteogenic sarcoma. Moreover, most osteogenic sarcomata involve both the medullary cavity and the periosteal region.[8]

Important, but not diagnostic, is the Codman triangle. This represents perpendicular striations of new bone arising from the periosteum, which has been elevated by and reacts to tumor tissue. The triangle derives its name from the acute angle of cortex with elevated periosteum. The idea of the triangle is to dramatize the reaction of the lifted periosteum and to indicate that only one side of the bone may show this change. Its presence is not essential for the diagnosis. It will be found more frequently, however, if oblique views, as well as the usual anteroposterior and lateral views, are taken, for then periosteal reaction will be found at the perforation of the bone by the tumor.

PATHOLOGY

GROSS

Usually, the tumor is found to originate in the metaphysis. When the epiphysial plate is open, the cartilage acts as a barrier to the tumor. When epiphysial cartilage is closed, the tumor then may extend into the epiphysial end of the bone (Fig. 2).

Invasion by the tumor from its primary metaphysial location occurs in 3 ways:

First, through the cortex with resultant periosteal lifting. Then, if the periosteal barrier is broken, the tumor invades the soft tissues about the bone. In some instances, particularly when growth is rapid, the fundamental architecture of the cortex may appear relatively intact, with penetration occurring through the haversian systems. When growth is slower, expansion or erosion of the cortex takes place.

Second, medullary spread may occur. This medullary plug often is the most cellular portion of the tumor.[8] This conical tumor may



FIG. 2. Osteogenic sarcoma. Same patient as in Figure 1. Amputation specimen. Note the intact epiphysial plate but the wide expansion of the lesion in the distal metaphysis and the subperiosteal regions. This type of lesion often shows elevation of the serum alkaline phosphatase. (Dr. M. M. Pomeranz)

extend several inches and still not show destruction or ossification roentgenologically. It is important to bear this in mind in determining the level of amputation.[8]

The third, and least common, pathway for spread is to the epiphysial plate or across the closed plate into the epiphysial area. It does not cross the articular cartilage. Joint involvement occurs, but only as a result of soft tissue spread.

The texture of the tumor is entirely related to the proportion of new bone to cellular tissue. Sclerosing lesions may be as hard as compact bone, osteolytic lesions may be fibrous or even mushy in consistancy, especially in the presence of hemorrhage and/or necrosis. Of course, variations by combinations of the 2 above reactions may be found. Sometimes extreme vascularity or telangiectasis may be present.

MICROSCOPIC

Just as the gross picture may vary on the basis of duration of the growth, the presence or the absence of the epiphysial plate, or combinations of osteolytic and osteoblastic types, the microscopic picture also may vary markedly in different cases (Fig. 3).

One must consider the tumor to be one of the bone components themselves, namely, of osteoblasts and tumor osteoid. So, regardless of the variations, the characteristic findings may be summarized as follows:

1. This is a sarcomatous connective tissue stroma. This shows either large atypical spindle cells or cells that are very anaplastic and irregular in pattern. These latter may coalesce into tumor giant cells.

2. Tumor osteoid and tumor-bone formation of wild type accompanies this very cellular reaction. The osteoid, which may or may not be ossifying, appears to be surrounded by relatively small tumor osteoblasts. These cells appear to be less anaplastic than adjacent larger ones that are located in the fibrous portion of the tumor.

3. Areas of tumor cartilage formation are found in which there are irregular cells located within a cartilage matrix.

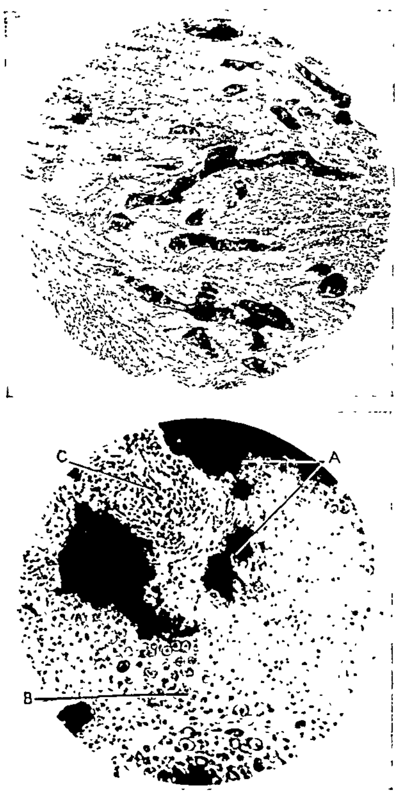


FIG. 3. (*Top*) Osteosarcoma with newly formed bone lamellae (× 35). (*Bottom*) Osteochondrosarcoma with calcified osteoid tissue, A; atypical cartilage cells, B; undifferentiated sarcoma tissue, C (× 80). (Ehrich, W. E.: Pathology for Students and Practitioners of Dentistry, Philadelphia, Lea & Febiger)

4. When there is osteoblastic or sclerosing bone formation, this bone incorporates and flows along the previous normal trabeculae. In the subperiosteal region, new tumor bone will be seen juxtaposed with new bone formed by periosteal reaction.

5. Microscopic changes that correspond to the gross areas of hemorrhage, necrosis, cyst formation and telangiectasia will be observed. These, however, are not diagnostic.

There have been well-documented reports of multicentric osteogenic sarcomas. In other words, primary osteogenic sarcoma has been reported to occur simultaneously in 2 or more widely separated areas. This is in contrast with the not uncommon osseous metas-

tases from a single primary osteogenic sarcoma.

Metastasis usually is by the hematogenous, not the lymphatic, route. Invariably the lungs become involved. Other viscera structures rarely are affected. However, extension to regional lymph nodes has been observed occasionally.

COMPLICATIONS

Local ones include pathologic fracture and soft tissue breakdown of a rapidly growing ulcerating tumor. Then secondary infection may well result.

Invasion of blood vessels with hemorrhage also occurs.

General. Rapid debilitation of the patient occurs with chest symptoms particularly prominent.

Biochemical Aspects. The only aberration in blood chemistry that may occur is elevation of the serum alkaline phosphatase from the activity of the tumor.[1,5,10] The thought was based upon the observation that the level decreased after ablation, excision or irradiation of the tumor.[3,11] Although this is interesting, and more data ought to be collected, correlation of alkaline phosphatase levels with degree of activity of the tumor cannot yet be made. Diminution of alkaline phosphatase following irradiation is probably the result of local destruction of this enzyme by radiation.

TREATMENT

Ablation as quickly as possible is the accepted current treatment. We agree with Lichtenstein that the 5-year survival period is not greater than 10 per cent. Earlier figures of larger numbers of survivors are felt to be based upon cases of chondrosarcoma included in the osteogenic sarcoma series.

Phemister reported a long survival of a case of osteogenic sarcoma treated by resection of the involved bone.

Goldenberg reported an 8-year survival in a case of osteogenic sarcoma. A mestastasis to the right upper lobe of the lung, which occurred 3 years after the original mid-thigh amputation, was treated by lobectomy without known recurrence after five years.[4]

X-ray and other forms of radiation are at best palliative. These tumors are markedly radioresistant.

REFERENCES

1. Bodansky, A., and Jaffe, H. L.: Phosphatase studies; serum phosphatase in disease of the bone: interpretation and significance, Arch. Int. Med. 54:88, 1934.
2. Coley, B. L.: Neoplasms of Bone, New York, Hoeber, 1949.
3. Franseen, C. C., and McLean, R.: Phosphatase activity of tissues and plasm in tumors of bone, Am. J. Cancer 24:299, 1935.
4. Goldenberg, R. R.: Eight year survival of osteogenic sarcoma of the tibia with pulmonary metastasis, Bull. Hosp. Joint Dis. 15:67, 1954.
5. Gutman, A. B., Tyson, T. L., and Gutman, E. B.: Serum calcium, inorganic phosphorus, and phosphatase activity in hyperparathyroidism, Paget's disease, multiple myeloma and neoplastic disease of the bones, Arch. Int. Med. 56:379, 1936.
6. Hatcher, C. H.: The development of sarcoma in bone subjected to roentgen or radium irradiation, J. Bone & Joint Surg. 27:179, 1945.
7. Jaffe, H. L.: Tumors of skeletal system; pathological aspects, Bull. New York Acad. Med. 23:497, 1947.
8. Lichtenstein, L.: Bone Tumors, St. Louis, Mosby, 1952.
9. Martland, H. S., and Humphries, R. E.: Osteogenic sarcoma in dial painters using luminous paint, Arch. Path. 7:406, 1929.
10. Stein, I., Stein, R. O., and Beller, M. L.: Living Bone in Health and Disease, Philadelphia, Lippincott, 1955.
11. Woodard, H. Q., and Higinbotham, N. L.: Correlation between serum phosphatase and roentgenographic type in bone disease, Am. J. Cancer 31: 221, 1937.

Sarcoma Osteogene del Osso

Summario in Interlingua

Sarcoma osteogene es le plus commun maligne tumor primari del osso. Su nomine indica que illo contine histos ossiformante, ben que de character anaplastic. Su culmine de frequentia occurre in adolescentes e

vene adultos, sed post le etate de 40 annos il ha ancora un gruppo considerabile in casos de morbo de Paget. Su etiologia non es cognoscite. Il pare que trauma ha un rolo connective in illo.

Symptomas es difficile a detegar durante le prime stadios, sed un persistente dolor o dolorositate debe esser interpretate como signal de urgentia pro le initiation de effortios diagnostic, e specialmente de studios radiographic. Le constatation cardinal in le roentgenogramma es destruction de osso. Associate con isto on nota un plus o minus marcate formation de osso nove, o del parte del tumor mesme o del parte del circumjacente osso e periosteoma. Le triangulo de Codman, que representa striationes perpendicular de osso nove emergente ab le periosteo elevate per le tumor, es importante sed non diagnostic. On distingue duo formas de sarcoma osteogene: le variante osteolytic e *le variante osteoblastic o sclerotisante.* Le prime forma pauc osso nove, le secunde multe.

Le lesion se manifesta usualmente in le metaphyse de un osso longe, specialmente in le region del genu o spatula.

Le diffusion local occurre in tres manieras:

1. A traverso le cortice, via le systemas haversian, a in le histos subperiosteal e molle circa le osso.

2. Dissemination medullar per un portion extrememente cellular del tumor. Iste area pote extender se ultra le radiographicamente detegibile portiones del crescentia, lo que frequentemente rende difficile le determination del grado de ablation.

3. Diffusion epiphysee occurre si le platta epiphysee es claudite. Usualmente le cartilagine articular non es violate, sed diffusion intra-articular pote occurrer via le histos molle que es invadite in le maniera de (1) supra.

Le criterios histologic depende del presentia de sarcomatose stroma de histos conjunctive ab le qual osteoid e osso tumoral se forma per metaplasia directe. Cartilagine tumoral es etiam trovate.

Metastase occurre via le venas al pulmones. Hematogene metastases ossee es etiam commun. In certe casos le origine es ossee e multicentric.

Le sol constatation hematochimic es le elevate phosphatase alcalin del sero. Isto pote abassar se post ablation o irradiation.

Le sol efficace therapia es ablation. Le superviventia cinquenne es probabilemente non plus que 10 pro cento. Iste tumores es marcatemente radio-resistente.

5

Primary "Round Cell" Sarcomas of Bone*

LAWRENCE J. MCCORMACK, M.D., HARRY B. ORRINGER,† M.D., AND GEORGE S. PHALEN, M.D.

Certain malignant bone tumors characterized by cells loosely termed *round cells* are of special interest to the orthopaedist, the pathologist and the radiologist. Not too many years ago, *round cell sarcoma* was considered to be a satisfactory diagnostic term. It has become apparent, however, that not all *round cell* sarcomas possess the same biologic behavior; and we now recognize at least 3 separate types of malignant bone tumors within this general group. The categories are Ewing's sarcoma, reticulum cell sarcoma, and plasmacytoma.

Although the final diagnosis of these bone lesions is the responsibility of the orthopaedist, he requires close co-operation from the radiologist and from the pathologist. Clinical and roentgenographic findings sometimes are diagnostic of bone tumor, but they are not substitutes for histopathologic findings. The pathologist must have a good working knowledge of the general clinical and roentgenographic characteristics of bone lesions, and of course he must exercise meticulous care in the handling of all tissue submitted to him lest he negate the efforts of the other specialists involved.

Notwithstanding the lack of evidence that biopsy ever has been responsible for the dissemination of any neoplasm,[2] we believe that dissemination does occur as a result of manipulation of the tumor. This statement, however, must be tempered by the recognition that the activity of the patient's muscles, the palpating hands of the physician and the pressures exerted at the time of surgery all can be implicated in the problem of venous dissemination. We have come to this conclusion on the basis of our own studies of venous blood draining from various systemic malignant neoplasms, as well as of the studies of Engell[3] concerning this mode of dissemination. As a consequence, the dangers of biopsy become relatively less significant, as accurate diagnosis is the best basis for adequate therapy.

The morphologic diagnosis of a bone tumor is accomplished best if certain rather rigid rules are followed. A specimen removed by the surgeon always should be screened by frozen section technic. It is indeed a rare tumor in which there is so much osseous material that a small soft portion cannot be removed for study. Although at times the pathologist cannot give a definitive diagnosis, at least he can attest to the adequacy of the specimen—a most important consideration. He also has an opportunity, if he so desires, to perform other important tests. Not all destructive lesions of bone are neoplastic; the recognition of the inflammatory nature of a lesion can afford the necessary arrangements for cultural study of any bacteria that may be present.[6] Those who dislike or dis-

* From the Departments of Pathology and of Orthopedic Surgery, The Cleveland Clinic Foundation, and The Frank E Bunts Educational Institute, Cleveland, Ohio.

† Fellow in the Department of Orthopedic Surgery, Cleveland Clinic

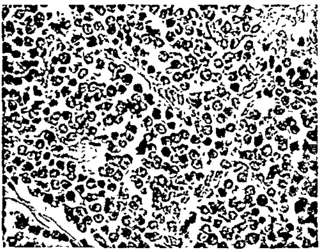


FIG. 1. Ewing's sarcoma. Photomicrograph demonstrating uniform cell pattern and some tendency toward compartmentation by small septa of fibrous tissue. Hematoxylin-eosin, × 450

approve of frozen section in the study of bone neoplasms either have had no experience with the method or have been the unfortunate victims of poor technic. Regretfully we admit that at the present time there is no method of decalcification that produces uniformly successful results. We have found it best to select the least bony portions of the surgical specimen for fixation in modified Zenker's solution. In this manner the histologic detail is well preserved, and excellent sections can be prepared. We believe that one of the common causes of inaccurate diagnosis of a bone lesion is poor technical preparation.

EWING'S SARCOMA

This group of bone tumors first was segregated by James Ewing in 1921.[4] Believing that they were vascular in nature, he suggested the term *diffuse endothelioma* for the group. Unfortunately, others have not been so successful in demonstrating the possibilities of their being of angiomatous origin, and, as a consequence, since no site of origin has been proved, the eponym *Ewing's sarcoma* remains today one of the few eponyms recognized in nomenclatures of disease.

Clinically, the disease is one of young people, and it affects males slightly more frequently than females.[7] The most common sites of the lesion are femur, tibia and humerus. The lesion is most prevalent in the second decade of life; rarely is it found in patients more than 30 years of age. In addition to the symptoms of pain and the findings of heat and swelling, there may be evidences of a systemic reaction in the form of fever and leukocytosis. These clinical findings may lead to a tentative diagnosis of osteomyelitis. If antibiotic or chemotherapeutic agents are administered, the lack of response to them should suggest the possibility that the lesion is not inflammatory.

The roentgenographic findings often are not characteristic. Bone destruction in the metaphyses of a long bone, especially of the lower extremities, is the most common feature. An extra-osseous mass usually is present, and the central portions of the involved bone may have mottling. The production of the so-called *onion peel* (laminations of periosteal reaction) has been overemphasized and rarely is found. At times, there may be seen some *sun-ray* radial striations that closely mimic the osteogenic sarcoma.

The histopathologic findings are striking in the uniform appearance of small closely packed round cells without cytoplasm, or with only a sparse amount, and a round nucleus containing finely divided chromatin (Fig. 1). Nucleoli are present rarely, but mitoses are frequent Unfortunately, large areas of the tumor may become necrotic, and often there is only perithelial sparing of tumor cells. This change results in islands of these cells surrounding a well-defined small vessel, imparting a perithelial pattern. The vessels in these tumors always are separate and distinct, and there is no evidence of the tumor cells forming vascular spaces. Clefting, which may be misinterpreted as a malformed vascular space, often is present as a shrinkage artefact. Thrombi of tumor cells within adjacent small vessels can be seen commonly.

Three other bone tumors especially can

mimic closely, and at times cannot be separated from, Ewing's sarcoma. Metastatic neuroblastoma can simulate completely the Ewing's sarcoma, both clinically and morphologically. In our experience, however, there has been some tendency for the cells to be much more darker staining and to show more nuclear irregularity than is seen in the Ewing's sarcoma; pseudorosettes are rare. The demonstration of these distinguishing features, of course, rests upon meticulous histologic technic. Localized leukemic infiltrations can give one occasional difficulty in so far as the histologic material is concerned. If one persists in attempting to diagnose the lesion in older age groups, the small cell bronchogenic carcinomas will pose a most difficult histologic problem, and often they cannot be distinguished from the lesion on morphologic grounds alone.

Proper therapy should be graded. Initially, following diagnosis, the patient should receive an adequate dose of irradiation. This treatment should be applied to the entire bone if at all possible, as spread within the marrow cavity can occur without giving evidence on the roentgenogram. Following this treatment, the patient should be watched carefully for any evidences of further tumor activity. If the lesion progresses, the only course is to amputate the limb. The prognosis in this particular group of neoplasms undoubtedly is poor. In series reported in recent years, salvage for long periods of time is effected in 10 per cent or less of the patients.

PRIMARY RETICULUM CELL SARCOMA OF BONE

In 1939, Parker and Jackson[9] segregated this group accurately as a neoplastic entity, and the category has been accepted for inclusion in most nomenclatures of bone tumors. The biologic behavior of reticulum cell sarcoma of bone distinguishes it as a separate, distinct entity; however, in a rare case there is a gray zone in which reticulum cell sarcoma is fused with Ewing's sarcoma, or the bone manifestations may be only one phase of a generalized disease that is histologically indistinguishable from the solitary lesions of bone. Additionally, a markedly dedifferentiated plasma cell myeloma also can cause a great deal of difficulty in differential histologic diagnosis. This mingling and overlapping of various neoplasms of marrow at times complicates the diagnosis.

Clinically, although the lesion can attack

FINDINGS IN 11 PATIENTS HAVING RETICULUM CELL SARCOMA

Case No	Age	Sex	Site of Tumor	Duration of Symptoms Before Treatment	Comments
1	32	M	Upper tibia	6 mo.	Living and well: 13 yr.; above knee amputation
2	34	M	Ilium	3 mo.	Dead: extradural extension with transverse myelopathy
3	38	M	Skull	?	Living and well: 7 yr.
4	39	F	Lower femur	1½ yr.	Lost to follow-up
5	41	M	Lower tibia	2 yr.	Living and well: 10 yr.
6	43	M	Upper tibia	11 mo	Under treatment
7	51	F	Thoracic spine—10th	5 mo.	Dead: 5 mo.
8	54	F	Lumbar—3rd	6 mo.	Dead: 9 mo.
9	63	F	Ilium	12 mo.	Dead: 4 yr.
10	63	M	Ischium	6 mo.	Lost to follow-up
11	64	F	Ilium	5 mo.	Living and well: 5 yr.

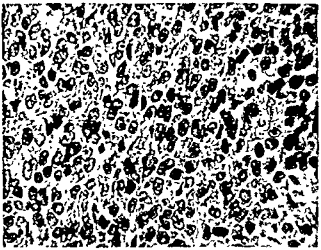


FIG. 2. Reticulum cell sarcoma (Case 2). Cells are similar in type to oval or lobulated nuclei. Mitoses are frequent. Hematoxylin-eosin, × 450.

any bone, it occurs most frequently in the metaphyses of the long bones. The tumor develops in an older age group than does Ewing's sarcoma, the average age of those having reticulum cell sarcoma being a decade or more greater. The incidence of the lesion is 3 times more frequent in men than in women However, in the small group described in the table on page 49, there are almost as many women as men.

The signs and the symptoms of the disease are for the most part nonspecific. There is some tendency for the patients with the disease to have a more protracted clinical course with milder symptoms than do patients with other bone lesions. Pain and a gradually enlarging mass are common, and pathologic fracture occurs frequently. One clinical feature that may be of diagnostic value is that the lesion can be very large by the time the patient consults a physician, and yet he appears to be in good health. The laboratory findings are of little value; the one most commonly positive is the elevation of the sedimentation rate, a nonspecific finding.

Roentgenographic findings also are nonspecific. The chief effect of the tumor is lysis of bone with resultant destruction of the shaft and mottling of the remaining bone. A soft tissue mass associated with the lesion is seen commonly.

The gross pathologic features are not specific. In study of amputated tumors, 3 types of lesions generally are found. Occasionally one encounters a relatively small tumor that is limited entirely to the narrow cavity and may invade the adjacent epiphysis. A pyriform type of tumor mass, equally involving the shaft of the long bone and expanding into the surrounding soft tissue, also is encountered occasionally. Most commonly, the tumor mass will project from one bony surface as an eccentric, soft, friable gray or pink mass. Zones of necrosis frequently are seen, and the entire central portion of the tumor may be necrotic.

The histopathologic findings are specific only in delineating the lesion as a reticulum cell sarcoma, and clinicopathologic correlation is mandatory to determine whether or not the lesion may be classified as primary in bone. Two general cell types are seen:[8]

In one, the less common of the 2 types, the neoplasm is composed entirely of cells possessing a relatively large lobular and vesicular nucleus; cell size is relatively uniform. The cytoplasm is relatively sparse, and it may completely ring the nucleus uniformly, or at times be elongated, suggesting ameboid motion (Fig. 2).

In the second group, there appears to be an admixture of cell types, mimicking all the stages of lymphocytic development (Fig 3, *right*). The reticulum cells previously described are seen and have a tendency to occur in small groups outlined by fine collagenous bands. Surrounding these cells are slightly smaller cells that still have vesicular nuclei, suggesting lymphoblasts; and, finally, a cytologic element indistinguishable from the mature lymphocyte is a prominent feature.

The tendency of the neoplasm to invade the wall of supporting blood vessels often is of great aid in diagnosis. However, this feature is common to reticulum cell lymphomas elsewhere in the body. The peripheral

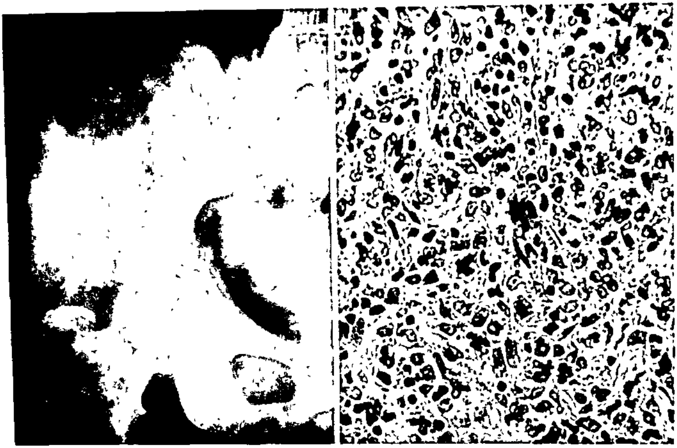


FIG. 3. Reticulum-cell sarcoma (Case 11 in table on page 49). (*Left*) Roentgenogram demonstrating mottled partial destruction of right ilium. (*Right*) Photomicrograph showing admixture of varying cell types: some are lobular or round and vesicular; others are small and resemble lymphocytes. Hematoxylin-eosin, × 450.

margins of the tumor invade surrounding tissues much as do the reticulum cell sarcomas anywhere within the body; for example, strands of the tumor can be seen coursing between individual fat cells. The neoplasm sometimes invades the adjacent regional lymph nodes. In a study of excised specimens, as many as 20 per cent show such extension.

Since the appearance of Parker and Jackson's[9] original article, judgments concerning therapy have changed. In their series, roentgen therapy was considered to be of relatively little value, but its efficacy has since been demonstrated. Initially, roentgen therapy is the treatment of choice in these lesions. If no response occurs, amputation should be resorted to in an attempt to eradicate the tumor. As a corollary, as much as possible of the nodal draining area also should be removed. The following case demonstrates a good result of roentgen therapy.

Case 1. A 64-year-old white housewife was first examined at the Clinic on May 24, 1951. She said that she had had pain in the right inguinal region and a limp of the right leg for 6 months. There was no history of trauma.

Examination of the lower back revealed a list to the left. The right hip and buttock were prominent, with slight induration and increased warmth of the area. Pain was experienced on abduction of the right hip and with straight leg raising There was ileopsoas and adductor muscle spasm. The right iliac crest revealed a prominent mass anteriorly, with obliteration of the anterior superior iliac spine. Roentgenograms showed an ovoid defect, measuring 8 by 6 cm., in the right ilium. The cortex superiorly was ruptured, and the ilium above the acetabulum showed a spotty radiolucency (Fig. 3, *left*). Biopsy of the right ilium revealed a reticulum cell sarcoma.

From June 2 to 28, 1951, 3,000 r were administered to the right anterior ilium and

3,000 r to the right posterior ilium. A regression of the lesion resulted, with restoration of normal function and appearance of the involved part.

Five years after treatment, the patient is living and well, apparently without evidence of recurrence or metastases.

SOLITARY PLASMACYTOMA (PLASMA CELL MYELOMA)

Carson, Ackerman and Maltby,[1] in their recent study of a large series of cases of myeloma, presented an excellent dissertation

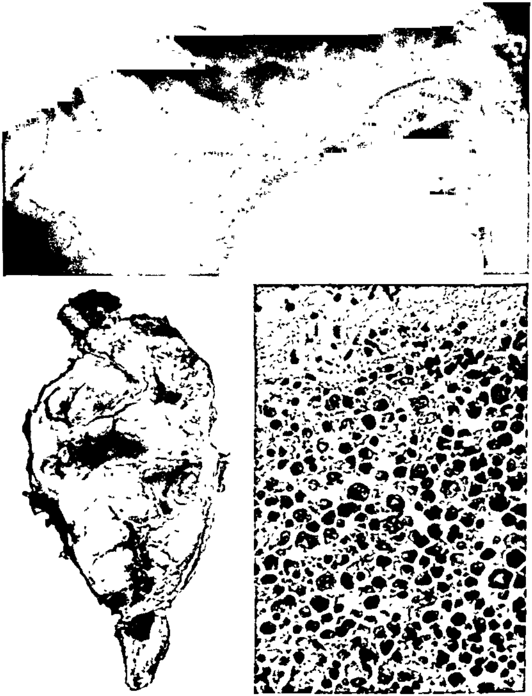


FIG 4 Apparent solitary plasmacytoma. (A, *top*) Roentgenogram demonstrating destruction of major portion of right clavicle. Some "bubbling" of bone is noted (B, *left*) Gross specimen of removed clavicle. (C, *right*) Bizarre myeloma cellular forms are closely packed Multinucleate forms and prominent hofs are present. The acellular material at top is amyloid. Photomicrograph, hematoxylin-eosin, × 450.

on the rare lesions, solitary plasmacytomas. They chose the term *apparent solitary plasma cell tumor*, stressing the fact that the vast majority of the patients finally developed disseminated plasma cell myeloma. We have seen 1 solitary plasmacytoma, previously reported,[5] that did not disseminate until approximately 6 years after it was diagnosed initially:

Case 2. A 46-year-old Negro housewife was first examined on August 2, 1946. She complained of a swelling in the right upper chest and shoulder and recurrent dull pains in the right shoulder that radiated up the right side of the neck and terminated behind the right ear. There was no history of trauma. Pain first was experienced 3 years previously; however, the mass first was recognized 1 year prior to admission to the Clinic, and it had become progressively larger.

Roentgenograms demonstrated a destructive lesion that involved the medial two thirds of the clavicle and was surrounded by an expansile soft tissue mass (Fig. 4A).

On August 20, 1946, a frozen section examination and excision of the proximal two thirds of the right clavicle were performed. The neoplasm measured 12 by 6 by 6 cm. and weighed 250 Gm. The mass was firm and protruded from the medial aspect of the clavicle; it extended inferiorly to the third rib anteriorly, medially to the mid-line, and superiorly to the level of the suprasternal notch (Fig. 4B). It was fixed to the clavicle and anterior chest wall. The histopathologic diagnosis was plasmacytoma with amyloid deposition involving the clavicle and adjoining soft tissue structures (Fig. 4C).

On September 4, 1946, a skeletal survey failed to reveal any other neoplasm. Other than a mild secondary anemia, laboratory findings were normal, including serum proteins, bone-marrow studies, urinalysis for Bence Jones proteins, sedimentation rate and routine blood studies.

On May 9, 1952, 6 years after initial examination, a roentgenogram showed evidence of an expanding lesion of the left 6th rib. Sternal puncture revealed an increase in the plasma cells with moderate immaturity.

On June 26, 1952, 4 inches of the involved 6th rib was excised. The pathologic diagnosis was plasmacytoma. Deep roentgenotherapy was given, 3,000 r to the anterior chest from June 30, 1952, to July 16, 1952. The patient was asymptomatic until the first week of April, 1954, when she noticed a dull pain and swelling in the left chest over the site of previous surgery and above the left breast. The area was irradiated with 3,000 r. In addition, the patient was given enseals of urethane.

By March 14, 1955, the mass above the left breast had disappeared, and the patient apparently was in good health. At that time, the serum proteins were increased slightly, 7.43 Gm./100 ml.; the gamma globulin was increased appreciably, 1.52 Gm./100 ml.; and the sedimentation time was elevated, 1.6 mm./minute.

The patient was seen last on October 13, 1955. She was taking urethane enseals (0.3 Gm. 6 times daily) and was asymptomatic and in apparent good health.

Grossly, the appearance of these tumors is not distinctive, being merely a destructive lesion of bone, usually producing an associated soft tissue mass. The histopathologic findings in the plasmacytoma are identical with those in the plasma cell myeloma; the tumorous plasma cell (myeloma cell) possesses a wide variation. It may be indistinguishable from a mature plasma cell, or it may be indistinguishable at times on histologic preparation from the reticulum cell group. Bone-marrow studies are of aid in differentiation.

SUMMARY AND CONCLUSIONS

The differential diagnosis of *round cell* sarcomas of bone has been discussed. Actually, these tumors are separable into several distinct types of bone neoplasms. Apparently, Ewing's sarcoma and primary reticulum cell sarcoma often are solitary bone lesions. The solitary plasmacytoma and the rare lymphocytic lymphosarcoma of bone probably are not solitary lesions; generalized disease usually will develop, but sometimes it requires years.

Careful co-operation among surgeon, pathologist and roentgenologist, utilizing every diagnostic tool, including the clinical history, roentgenogram, frozen section diagnosis and excellent permanent sections, will resolve the problem in practically every case.

A correct diagnosis is mandatory, as therapy is guided by the cell type of the neoplasm.

REFERENCES

1. Carson, C. R., Ackerman, L. V., and Maltby, J. D.: *Plasma cell myeloma. A clinical, pathologic and roentgenologic review of 90 cases.* Am. J. Clin. Path. 25: 849, 1955.
2. Dahlin, D. C.: Editorial—Bone tumors: some aspects of their diagnosis, Am. J. Clin. Path. 25:935, 1955.
3. Engell, H. C.: Cancer cells in the circulating blood, Acta chir. scandinav., Supp. 201, p. 70, 1955.
4. Ewing, J.: Diffuse endothelioma of bone, Proc. New York Path. Soc. 21:17 and 23, 1921.
5. Jones, T. E., and Hazard, J. B.: Plasmocytoma of clavicle, Cleveland Clin. Quart. 14: 198, 1947.
6. McCormack, L. J., Dickson, J. A., and Reich, A. R.: Actinomycosis of the humerus, J. Bone & Joint Surg. 36A:1255, 1954.
7. McCormack, L. J., Dockerty, M. B., and Ghormley, R. R.: Ewing's sarcoma, Cancer 5:85, 1952.
8. McCormack, L. J., Ivins, J. C., Dahlin, D. C., and Johnson, E. W., Jr.: Primary reticulum-cell sarcoma of bone, Cancer 5:1182, 1952.
9. Parker, F., Jr., and Jackson, H., Jr.: Primary reticulum cell sarcoma of bone, Surg., Gynec. & Obst. 68:45, 1939.

Primari Sarcomas Ossee a Cellulas Ronde

Summario in Interlingua

Primari sarcomas ossee a "cellulas ronde" se classifica in tres typos: (1) Sarcoma de Ewing, (2) sarcoma a cellulas reticulari, e (3) plasmocytoma. Un stricte cooperation inter orthopedista, pathologo, e roentgenologo es necessari pro establir le diagnose e planar le curso therapeutic.

Le *sarcoma de Ewing es un morbo de* personas juvene. Le sitos preferite es le femore, le tibia, e le humero. In ultra de tumescentia, dolor, e dysfunctionamento, il occurre a vices un reaction systemic que se manifesta per febre e leucocytosis. Le tractamento initial es irradiation del integre osso involvite. Sed si le neoplasma avantia, am*putation debe esser prendite in consideration.* Le prognose non es bon.

Sarcoma a cellulas reticulari occurre in personas de etates plus avantiate que sarcoma de Ewing. Omne osso pote esser afficite, sed le sito de preferentia es le metaphyses del ossos longe. Le signos e symptomas es frequentemente leve. Illos include dolor e un massa a crescentia gradual. Fracturas pathologic pote occurrer. Osteolyse con destruction partial del diaphyse e marmorisation del resto del osso es un constatation commun. Le tractamento de election es roentgenotherapia. Sed si illo remane sin successo, amputation debe esser prendite in consideration. Inter le plus commun primari neoplasmas ossee, sarcoma a cellulas reticulari ha le melior prognose, si le supramentionate tractamento es usate.

Plasmocytoma "solitari" del osso es un lesion rar que occurre al medietate del vita e postea In le majoritate del casos, un disseminate myeloma a cellulas plasmatic se disveloppa in le curso del tempore. Le tractamento a initiar es roentgenotherapia. Si illo non succede o si il ha symptomas de compression, intervention chirurgic es a recommendar.

6

Primary Round Cell Tumors of Bone—Some Problems Concerning Their Management*

JOHN H. WALKER, M.D.,† AND HUGH W. JONES, M.D.†

The term *round cell tumor* is employed here to designate certain neoplasms arising in bone whose histogenesis is obscure or unknown. This descriptive term is utilized as a working diagnosis for the highly cellular, undifferentiated or partially differentiated tumors. It serves a useful purpose by offering the surgeon or the radiologist a positive therapeutic approach, bracketing a group of tumors in which aggressive roentgen therapy may achieve unexpected long-term salvage.

A problem frequently arises in establishing an unequivocal histologic diagnosis in nonosteogenic bone tumors when a variant or atypical tumor of the round cell group is encountered. The tumors in this group are: Ewing's tumor; primary reticulum cell sarcoma of bone; and myeloma. A brief review of these tumors emphasizes certain common features.

Ewing's Tumor. Ewing's tumor, or sarcoma, is a controversial tumor. Its histogenesis is unknown. Some oncologists have denied that it is an entity, suggesting that it may be metastatic neuroblastoma or a catch-all diagnosis for a number of unrecognized tumors. The criteria for a diagnosis often have included clinical and roentgenologic *confirmation*. The patients are in the younger age group; 85 per cent or more of recorded cases are less than 30 years of age. The roentgenologic picture was thought originally to be typical, but experience over the years has shown that Ewing's tumor is a great imitator of bone pathology. Microscopically it may be recognized by sharply defined criteria often obscured by degeneration or atypical forms.

Reticulum Cell Sarcoma. Primary reticulum cell sarcoma of bone is an entity to be differentiated from generalized reticulum cell sarcoma, Ewing's tumor or myeloma. The criteria for recognition include a primary focus of tumor in a single bone histologically typical of reticulum cell sarcoma without initial evidence of disseminated tumor. The age spread is broader than in Ewing's tumor, and it occurs frequently in older individuals. The microscopic diagnosis is definitive when typical. Inflammatory lesions with histiocytic response, atypical Ewing's sarcoma and myeloma have been obstacles in accurate diagnosis.

Plasma Cell Myeloma. The term *solitary plasma cell myeloma* has been employed for plasma cell tumor involving a single focus in bone. This is a combined diagnosis by roentgenographic and marrow study exclusions of multiple or diffuse tumor. In 1950, Christopherson and Miller[4] reviewed the cases of solitary myeloma in the literature and concluded that it was unrelated to multiple myeloma, and that the prognosis was uniformly good. Others, notably Carson *et al.*,[3] consider that the bulk of these are early

---

* Permission to quote from a previous paper by the authors, "The Irradiation Management of Primary Round Cell Tumors of Bone," has been kindly granted by the editors of *Radiology*. This paper appeared in *Radiology*, vol. 61, pp. 738 to 748, 1953.

† From the Departments of Radiology and Pathology, The Mason Clinic, Seattle, Wash.

lesions of the disseminated myeloma with a long period of focal growth. Accurate histologic diagnosis of the tumor by aspiration or biopsy is possible in the majority of cases. Chronic infection with plasma cell response, bizarre changes in plasma cell morphology or degeneration in the tumor may lead to error in individual cases.

It is significant that:

1. Where round cell tumors have been re-evaluated critically, Ewing's sarcoma frequently has been an erroneous diagnosis.
2. Primary reticulum cell sarcoma of bone seldom is listed in large series as an entity, but, when recognized, the prognosis has been uniformly good. The tumor deserves particular attention as a malignant, nonosteogenic round cell tumor of bone which, even though involving multiple bones and anatomically inoperable, should receive aggressive rather than palliative radiation.
3. Myelomas appearing as solitary tumors may closely resemble Ewing's tumor histologically. This has been noted by Johnson[12] and others. These tumors are amenable to intensive radiation.

It is particularly noteworthy that a substantial percentage of final diagnoses of *round cell tumors is made in hindsight.* A recent appraisal of the results of radiation therapy in a group of bone tumors treated at The Mason Clinic made it apparent that the proportion of survivals in patients having Ewing's tumor was high compared with larger, well-documented series. The original diagnosis of Ewing's tumor was established on the basis of the clinical history and roentgenographic evidence and confirmed by adequate biopsy. With renewed interest, blocks of tumor tissue were recut and made available for examination by several reputable pathologists.* Four of the cases in our series, which were thought at one time to represent Ewing's tumor, were reclassified. In 3 of these, the final accepted diagnosis was reticulum cell sarcoma and 1 solitary myeloma. Our experience is not unique. Jackson and Parker,[11] reporting 25 cases of reticulum cell sarcoma, primary in bone, found 13 which had been classified previously as Ewing's sarcoma in the Bone Registry of the American College of Surgeons. In the collection of 37 cases of primary reticulum cell sarcoma, Coley *et al.*[5] found that 7 were interpreted originally as Ewing's sarcoma. Prebo[16] lists 55 cases of Ewing's tumor in an analysis of 205 cases of malignant primary bone tumors. He makes no mention of reticulum cell sarcoma. Rosh,[17] in a report of 121 treated cases, omits any reference to reticulum cell sarcoma The entity of reticulum sarcoma, although well established in 1939, seldom is mentioned in the orthopaedic literature.[6]

The primary consideration when evaluating a lesion of bone is the segregation of bone tumors from other nonneoplastic processes. Clinical symptomatology, usually an important link in establishing specific diagnosis, is of little value here. The presenting symptom of pain, accompanied by fever and *localized swelling, may be present in* bone tumor or osteomyelitis. The physician confronted with this triad may delay diagnosis by prolonged antibiotic therapy. Radiologists and orthopaedists recognize the pitfalls in roentgenographic diagnosis of bone tumors, and these difficulties need not be reemphasized. No clear-cut diagnostic differentiation can be made as to the cell type of any particular osteolytic tumor of bone by roentgenographic means.[1] Broad generalizations can be made as to the characteristics of certain tumors which may not be valid when applied to a single case.

The response of the tumor to irradiation as a diagnostic test is to be condemned. Once irradiation has been initiated, irreversible changes occur in the tumor and add further confusion to a situation already complex to the pathologist. It is unfortunate

* We are indebted to Fred W. Stewart, M.D., Memorial Center, New York, and to Lent C. Johnson, M.D, and Brigadier General Elbert DeCoursey, of the Armed Forces Institute of Pathology, for their review of our material and constructive comments

that the *therapeutic trial* of irradiation still is employed by many, and even advocated as a substitute for biopsy.[9,17] The diagnosis of round cell tumor of bone cannot be made on the basis of clinical symptoms or roentgen findings, and certainly not on the basis of the *therapeutic trial*. Specific diagnosis depends upon adequate histopathologic examination.

In our experience, biopsy offers no hazard to the patient's well-being or in the dispersal of tumor beyond its confines.[1,2] In each case illustrated here, treatment was begun only after study of permanent sections following open biopsy. Frozen sections are employed routinely as a guide for the surgeon to determine the adequacy of biopsy. The consultation of a pathologist and a radiologist were invaluable at the time of biopsy in obtaining representative sections from the tumor site. Where a specific diagnosis of the tumor was possible by recognizable and well-documented differential characteristics, treatment was instituted without the necessity of a general classification. Not infrequently, however, only generalizations and a working diagnosis of *round cell tumor* could be made from partially necrotic tissue from a rapidly growing lesion. This working diagnosis was utilized for immediate guidance and future management with the least procrastination.

An attitude of pessimism long has veiled the management of bone tumors, regardless of whether the treatment is by radiation or surgery or by combinations of both. This has been especially true of inaccessible lesions, as of the spine. The outlook has been considered hopeless by some observers when regional lymph nodes beyond the site of tumor have been involved. Curative rather than palliative efforts should be undertaken, even in those cases in which more than one bone is involved by tumor of this nature. Despite relatively homeopathic doses of irradiation, long remissions and apparent cures have been reported in the literature in isolated cases.[13] This is true of both reticulum cell sarcoma and the condition known as solitary myeloma. It is of interest to note that in two of the cases reported by Coley *et al.*,[5] in which recurrence developed after 10 years, small tumor dosage by present-day standards was employed. It seems probable that 5-year survivals will be reported more frequently by those subscribing to intensive judicious external irradiation (our Cases 3, 4 and 5). The general consensus manifested by the larger treatment centers throughout the country is that Ewing's tumor first should receive preoperative radiation followed by surgical amputation well above the lesion with postoperative radiation if indicated by the presence of positive lymph nodes unsuspected at the time of surgery. The 5-year survival in Ewing's tumor varies from nil[14] to 19 per cent.[8] Lichtenstein[14] attributes this discrepancy in statistics to improper identification of tumor. McCormack[15] reports that variation between those treated by surgery and by irradiation is so slight that it is not significant statistically. He then raises the question, "Are the 1-legged deaths worth the 1-legged cures?" Since reported long-term survivals are not significantly greater by one method of treatment than by another, perhaps the entire subject should be reappraised. We are convinced that many of the *cures* of Ewing's tumor by either method of treatment actually represent cures of reticulum cell sarcoma. Such being the case, it is possible that those patients with radical amputation would have survived without the amputation.

The results reported on sufficiently large series of reticulum cell sarcoma indicate without doubt that the treatment of choice is irradiation. There is not sufficient evidence at hand to support the concept that amputation either alone or in conjunction with irradiation offers the greater chance of survival.

Empirical formulas cannot be adopted or set forth for the irradiation management in malignant lesions of bone in the round cell group. Neverthless, certain precepts of conservative radiation should be followed. When



FIG. 1. (*Left*) Shows the complete loss of the architecture of the 1st lumbar, left transverse process, and the compression of the 4th thoracic body. There also is destruction of the pedicle and the spinous process of the 1st lumbar. (*Right*) Illustrates the reossification of the 1st lumbar, transverse process, pedicle and spinous process, 1 year following treatment. (Walker & Jones: Radiology 61: 738-748)

the primary site of tumor is in an extremity, then the entire bone should be subjected to irradiation. This should include an area well below the distal and above the proximal joints. Irradiation may be directed through opposing fields on alternating days covering the entire portion of the extremity under consideration If certain tumors prove to be unusually radiosensitive, the maximum tolerated dose of irradiation should be administered despite an unexpected favorable response. Faced with the apparent insurmountable odds of a large tumor of a large extremity and obviously involved regional lymph nodes, it is easy to fall into the trap of palliation. Our case No. 2 illustrates that intensive therapy carried out over a long period of time may produce an unexpected salvage. Where an extremity lesion is complicated by involved regional nodes, it must be assumed that there is similar invasion of deep pelvic and preaortic nodes; therefore, a plan of therapy to include these areas must be instituted. When the primary tumor involves more than 1 bone, such as in our Case No. 1, equal attention should be given each lesion. It is the routine practice in our department to check the adequacy of fields by suitable markers and follow-up roentgenograms. It is necessary to emphasize that the radiation fields or summation of the fields should be complete and encompass widely the lesion under treatment.

The following cases illustrate examples of unanticipated long-term survivals following radiation. The nonspecific term *round cell tumor of bone* was applicable in these cases in the preliminary diagnosis for instituting aggressive therapy. Further study, numerous sections with special stains and consultation established the final diagnosis given.

CASE SUMMARIES

Case 1. This 35-year-old housewife first was seen on March 26, 1947, with the complaint of back pain of 6 months' duration. The difficulty began in September, 1946, with sudden onset of pain in the upper back, between the shoulder blades. Since that time she had never been completely free of back pain, although it had migrated at intervals between the upper dorsal and the lumbodorsal areas In November, 1946, an area of swelling developed over the upper dorsal spine. This subsided after several weeks In January, a similar area appeared over the lumbodorsal spine. This subsided partially, but recurred.

Examination at the time of her first visit showed a well-developed young white woman in no acute distress. Over the lower dorsal spine was an area of tumefaction measuring 4 x 3 cm. This was firm and tender. Considerable limitation of motion was present in all planes. Neurologic findings were not significant, except for slight diminution of the left biceps reflex and an equivocal sensory loss on the inner aspect of



Fig. 3. (*Left*) Bone destruction along the cortex of the shaft with soft tissue calcification in the tumor itself. (*Right*) Postradiation roentgenogram demonstrates apparent areas of destruction within distal femoral shaft. This is the general picture 7 years after the treatment The areas of rarefaction actually represent segments previously occupied by tumor with secondary infarction and incomplete new bone formation. (Walker & Jones Radiology 61:738-748)

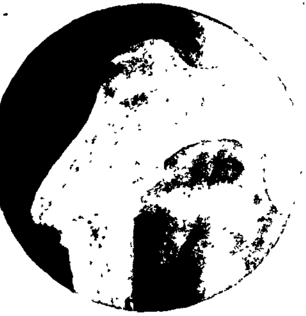


Fig. 4 Coned projection through plaster showing apparent union of the fracture through previously irradiated bone.

were treated on each day. The fact that large ports were used with relatively high dosage increased the irradiation intensity factor. As might be expected, the decrease in size of the tumor and the relief of pain were dramatic. The patient's well-being was manifest in ratio to the rapid disappearance of the tumor mass. After treatment of the lower extremity was completed, the patient was allowed to return home for a period of 2 weeks, with the provision that she

return for further irradiation. Opposing 10 x 20 cm. lower abdominal fields then were irradiated. These portals covered the lower presacral and preaortic chain. Irradiation first was administered at the rate of 250 r per day, alternating between anterior and posterior ports. This later was increased to 300 r per day. A total of 3,100 r measured in air was given to each. Follow-up roentgenograms showed deposition of calcium within the tumor mass itself; this, however, showed reabsorption gradually over the period of the next year. A definite increase in over-all texture and density of bone was the end-result. Multiple small areas of bone rarefaction still remained in the follow-up films, and show no change from year to year (Fig. 3, *right*).

The patient returned to full activity as an office secretary and has since married. Her only restriction of activities was the refraining from participation in athletic events. The lower extremity showed brawny induration and telangiectasia of the skin, but there was no restriction of motion.

In April, 1954, the patient inadvertently bumped her thigh, with a resultant fracture of the proximal third of the shaft. The fracture was through intensely irradiated bone, and, despite the opinion of several radiologists in her home town that the fracture was through recurrent tumor, it was our opinion that conservative management was in order. Withholding of biopsy was advised, as well as refraining from the use of an intramedullary rod. After a lapse of 1 year in hip spica, definite union was occurring. There was no evidence of recurrence or pain in either distal or regional areas. Edema of the lower extremity increased. The patient gained approximately 20 pounds because of her sedentary life.

At the present time there is no real indication of recurrent tumor. The development of union and the absence of pain are reassuring (Fig. 4).

Comment. This case illustrates a remarkable response to irradiation in a seemingly hopeless situation. It points up the precept of treating above and below the lesion and especially emphasizes intensive irradiation of the regional nodes as well as more distant nodes. This tumor, which was thought to have all the clinical and the roentgenographic characteristics of Ewing's sarcoma, has been reclassified since, and general agreement is that it is a reticulum cell sarcoma. The patient has survived 8 years since the onset of her disease. The occurrence of her *pathologic* fracture was not unanticipated as a postradiation sequela.

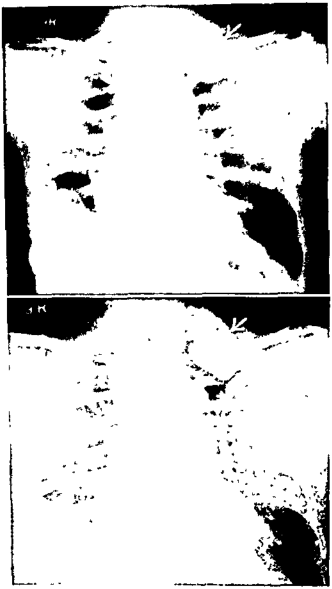


FIG. 5. (*Top*) Tumor completely replacing the left 1st rib, discovered in a mass chest roentgen survey. (*Bottom*) New bone formation within the grossly distorted rib several years after roentgen therapy. There has been no change in the appearance over a period of 6 years. (Walker & Jones: Radiology 61:738-748)

Case 3. This 55-year-old Syrian elevator operator stated that he was well except for slight discomfort in the left shoulder girdle, especially after a day's work, which he attributed to closing the heavy elevator door in a 40-story building. In a mass chest x-ray survey 7 years ago, a tumor that replaced completely the left first rib was discovered. The entire left rib was destroyed. However, the confines of the tumor were fairly



FIG. 6. (*Top*) Complete destruction of the 8th dorsal body. Only the superior and the inferior vertebral plates remain. The apparent bridging from the body of the T-7 to T-9 may have offered additional support when the 8th dorsal body collapsed. (Walker & Jones. Radiology 61: 738-748)



FIG. 7. (*Right*) A permanent kyphotic knuckle which developed over a 4-year period following completion of roentgen therapy. (Walker & Jones: Radiology 61: 738-748)

well outlined in the roentgenographic examination (Fig 5, *top*). The proximal half of the left clavicle was removed at time of surgical approach in an effort to resect the entire first rib and tumor mass. It was the opinion of the surgeon that resection was not feasible for obtaining a cure, and the patient, therefore, was referred back to the Department of Radiology for further management. Large opposing anterior and posterior ports, entirely encompassing the lesion and a sufficient portion of normal surrounding tissue, were employed The calculated tumor dosage was 3,820 r administered at the rate of 200 r per day. Response to irradiation was good. An extremely intense epidermite developed, with resultant telangiectasia and permanent atrophy of the skin.

Comment This case is of special interest in that the lesion had not caused sufficient symptoms for the patient to seek medical investigation It was uncovered incidentally as a result of a mass chest survey Originally diagnosed as a Ewing's sarcoma, the lesion has completely filled in with bone in a grossly distorted rib, but now has a definitely benign appearance (Fig. 5, *bottom*) The patient is well. He has no complaints and has returned to full occupation as an elevator operator. The partial resection of his left clavicle has caused him no disability. After review of permanent sections, the consensus is reticulum cell sarcoma. The patient has survived 7 years without recurrence.

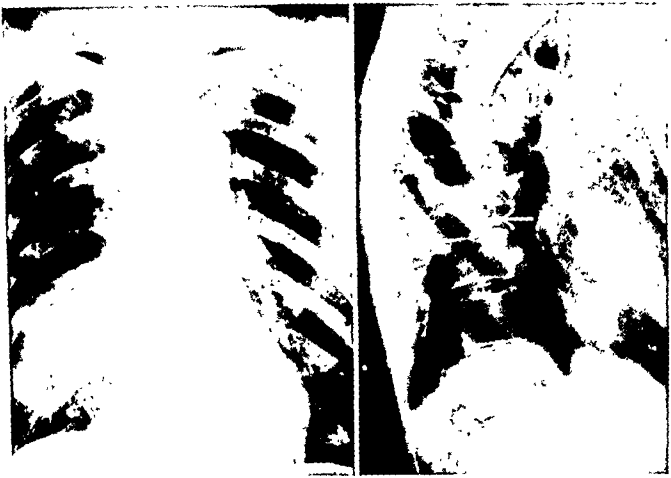


FIG. 8. Solitary myeloma. Note the extensive destruction of the right 9th rib with contiguous involvement of the 9th dorsal body with almost complete destruction. (Walker & Jones: Radiology 61:738-748)

Case 4. This 57-year-old white female stated that in the early 1930's her spine was fractured in an automobile accident but that recovery from this injury was uneventful. One year later her back again was injured in a fall. Again recovery was satisfactory. A third injury occurred in 1946 and was followed by a satisfactory response after 2 weeks of bed rest. Two months before examination there was a recurrence of backache that radiated to the lower ribs and, in addition, progressive difficulty in locomotion to the point of complete inability to walk and marked impairment of voluntary movements of the legs. Paraplegia developed rapidly. Roentgenographic examination showed complete collapse, both anteriorly and posteriorly, in the 8th dorsal body (Fig. 6). Lumbar puncture indicated a spinal fluid block, with increased protein in the fluid. Wassermann and colloidal gold tests on the spinal fluid and the blood were both negative.

A laminectomy with decompression was done on January 15, 1948. There was anterior extension of an extradural mass that invaded the entire pedicle of the 8th dorsal vertebra, especially on the right side. As much of the tumor was removed as was possible. Six days following surgery irradiation was initiated, adequate fields to cover the lesion being employed.

A calculated tumor dose of 2,750 r was administered in a relatively short time at the rate of 300 r, as measured in air, per day. Regarding the roentgenographic findings, at the time of initiation of therapy there was a definite soft tissue tumefaction posteriorly over the involved 8th dorsal body. This soft tissue tumefaction could be seen as a fusiform mass on the postero-anterior projection of the chest. Subsequently, over the next 2 years, this vertebra became completely fused to the body immediately below. There now has occurred a sharp kyphotic knuckle with firm fusion, as seen in the lateral [illegible]

with the help of a cane.

For a period of 6 years this patient survived without evidence of recurrent disease. She was able to take care of herself, living alone and doing all her household work. She suffered

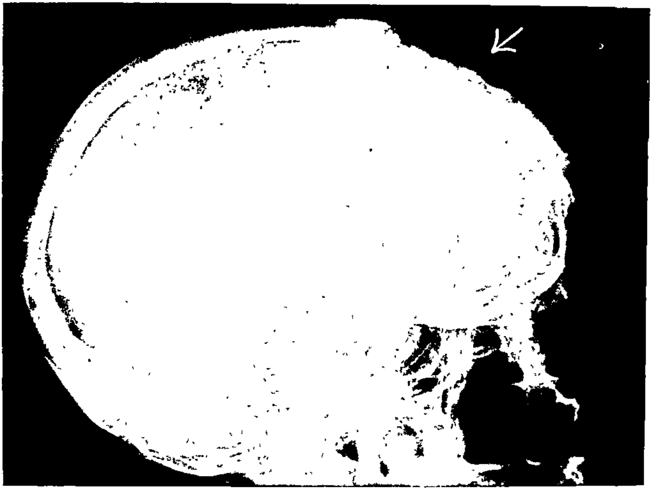


FIG. 9. Destructive lesion of the frontal bone. Biopsy. reticulum cell sarcoma.

minor radicular pain from time to time at the level of the 8th dorsal level and its corresponding dermatome. In August, 1954, she developed multiple metastases in almost all bones, quite typical of myeloma. She died in another hospital approximately 7 years after the initial diagnosis

Comment. This tumor was diagnosed originally as a round cell sarcoma, type undetermined. Subsequent more exhaustive studies have shown it to be plasma cell myeloma. Although the therapy administered was perhaps not as intensive as might have been given in retrospect, it was apparently sufficient to cause this 6-year remission without evidence of active disease until the 7th year, and then not at the site of original tumor.

Case 5. This 61-year-old white male was hospitalized on July 12, 1948, following recurrent back pain of 2 years' duration which had increased in severity during the past year. The pain was nonradiating and was aggravated by coughing and sneezing. He became completely bedridden 3 months prior to admission to hospital because of severe pain and some swelling over the lower dorsal spine, extending toward the right side at the level of the 9th rib.

Systemic review was negative. The roentgenographic examination showed destruction of the 9th dorsal vertebra (Fig. 8, *left*). His referring physician had reported a positive Bence-Jones protein in the urine on one occasion. A biopsy of the 9th rib was taken on the day following hospital admission. Biopsy was reported as either myeloma or Ewing's sarcoma. The lesions, which actually involved 2 bones but in direct continuity with each other, were treated through a single posterior field. The patient was extremely ill, and narcotics were required each day prior to his transfer to the Department of Radiology.

Pain relief following the beginning of irradiation was somewhat slower than in the other cases. He received a total of 4,305 r at the skin, including back scatter, at the rate of 200 r per day. The calculated tumor dose was approxi-

mately 2,400 r to the involved vertebra, and considerably higher in the rib.

Following completion of treatment the patient gained rapidly, was able to resume activity in his own laundry, and now does a full day's work.

Comment. Final diagnosis in this case was solitary myeloma. Although the tumor dose was not high, the patient has done well and now is in his 7th year of survival following onset of treatment. Of particular interest here is the reversal of the Bence-Jones protein in the urine; since the patient lives in Alaska, laboratory studies must be carried out there. His referring physician has not been able to detect the presence of Bence-Jones protein since his return there some 6 years ago.

Case 6. This 71-year-old white female was seen first on February 28, 1954, with a complaint of *growth* on the head. She noted a lump only 2 weeks before over the frontal area back of the hairline. There were no neurologic changes or other unusual physical findings other than a moderately indurated tumor mass diffusely involving the scalp and the frontal bone over an area of some 7 cm. on palpation.

Roentgenographic examination disclosed a destructive lesion in the posterior right frontal area measuring approximately 5 cm. in diameter and showing characteristics of malignant bone invasion. Both the outer and the inner tables were destroyed (Fig. 9). Tissue was obtained for histopathologic examination. Pathologic diagnosis was reticulum cell sarcoma.

Treatment was administered by external irradiation, 400 K.V., employing 2 opposing tangential fields of 10 x 10 cm each in the anterior and the posterior directions. Seven hundred fifty r, as measured in air, was directed posteriorly, and 1,550 r in air was directed anteriorly from the posterior port. In addition, 2 lateral fields of approximately 6 x 8 cm were utilized, delivering 1,500 r, as measured in air, in each. Immediate decrease in tumor size was apparent; however, there never has been complete filling in with new bone.

One year after completion of treatment, an enlarged upper cervical lymph node was noted on the right side. It was the opinion that this represented spread of the original disease. The referring physician removed the lymph node, which showed reticulum cell sarcoma.

The patient has since received irradation in her home town. At the time of her last visit to her local physician, she was well and there was no evidence of disease in the neck or persistent disease other than the defect in the skull.

Comment. The time interval since therapy has not been of sufficient duration to evaluate its efficacy; however, the sequence of events is that of primary bone reticulum cell sarcoma with regional metastasis, at present under control.

SUMMARY

Accurate classification of Ewing's sarcoma, reticulum cell sarcoma and solitary myeloma may be impossible at times. This problem is recognized with increasing frequency by those reviewing large series of bone tumors. Errors in diagnosis may result in the rendering of a relatively hopeless prognosis with attendant lack of aggressive therapeutic management. The *working diagnosis* of round cell tumor of bone in doubtful cases allows for an open mind in the prognosis and the application of therapeutic procedures. In most instances, irradiation is the preferred treatment because of the preservation of useful extremities in some and the inaccessibility to surgical approach in others. Illustrative cases are presented of unanticipated long-term survivals after irradiation.

REFERENCES

1. Brailsford, J. F.: The radiological evidence of malignancy in bone tumors and its relation to biopsy, Radiology 33:476, 1939.
2. ———: The serious limitations and erroneous indications of biopsy in the diagnosis of tumors of bone, Radiology 51:733-734, 1948.
3. Carson, C. D., Ackerman, L. V., and Maltby, J. D.: Plasma cell myeloma: a clinical, pathologic and roentgenologic review of ninety cases, Am. J. Clin. Path. 25:8, 849-888, 1955.
4. Christopherson, W. M , and Miller, A. J.: A re-evaluation of solitary plasma cell myeloma of bone, Cancer 3:240-252, 1950.
5. Coley, B. L., Higinbotham, N. L., and Groesbeck, H. P.: Primary reticulum-cell sarcoma of bone, Radiology 55:641-648, 1950.

6. Ewing, J.: Review of classification of bone tumors, Surg., Gynec. & Obst. 68:971, 1939.
7. Francis, K. C., Higinbotham, N. L., and Coley, B. L.: Primary reticulum cell sarcoma of bone, report of 44 cases, Surg., Gynec. & Obst. 99:143-146, 1954.
8. Geschickter, C. F., and Copeland, M. M.: Tumor of Bone, ed. 3, Philadelphia, Lippincott, 1949.
9. Henblein, G. W., Moolten, S. A., and Bell, J. C.: Some observations concerning Ewing's tumor, seen in an Army General Hospital, Am. J. Roentgenol. 56:688-706, 1946.
10. Ivins, J. C., and Dahlin, D. C.: Primary reticulum cell sarcoma of bone, J. Bone & Joint Surg. 35-A:835-842, 1953.
11. Jackson, J., Jr., and Parker, F.: Hodgkin's Disease and Allied Disorders, New York, Oxford, 1947.
12. Johnson, L. C., and Meador, G. E.: The nature of benign "solitary myeloma" of bone, Bull. Hosp. Joint Dis. 12:298-313, 1951.
13. Lawrence, K. B., and Lenson, N.: Reticulum cell sarcoma: report of a thirteen year survival following one thousand roentgens of x-ray therapy, J.A.M.A. 149:361-362, 1952.
14. Lichtenstein, L.: Bone Tumors, St. Louis, Mosby, 1952
15. McCormack, L. J., Dockerty, M. B., and Ghormley, R. K.: *Ewing's sarcoma, Cancer* 5:85-99, 1952.
16. Prebo, S. B.. A clinical analysis of 205 cases of malignant bone tumor, J. Bone & Joint Surg. 32-A:298-306, 1950.
17. Rosh, R., and Raider, L.: Primary malignant bone tumors: a review of cases seen in the radiation therapy department of Bellevue Hospital, Am. J. Roentgenol. 56: 75-83, 1946.
18. Sherman, R. S., and Snyder, R. E.: The roentgen appearance of primary reticulum cell sarcoma of bone, Am. J. Roentgenol 58:291-306, 1947
19. Valls, J · Ewing's sarcoma (abstract), J Bone & Joint Surg. 30-B:728, 1948.
20. Valls, J , Musculo, D., and Schajowicz, F · Reticulum cell sarcoma of bone, J. Bone & Joint Surg 34-B:588-598, 1952.

Primari Tumores Ossee a Cellulas Ronde: Alicun Problemas in Lor Manipulation

Summario in Interlingua

Le autores insiste de novo super le difficultate e le importantia del diagnose differential de (1) sarcoma de Ewing, (2) sarcoma ossee a cellulas reticulari, e (3) myeloma solitari. Iste problema es recognoscite de plus in plus frequentemente per investigatores concernite con grande series de tumores de osso. A causa del differentias del implicationes prognostic e possibilemente therapeutic, un diagnose erronee de sarcoma de Ewing pote resultar in le non-initiation de un aggressive therapia irradiational in casos in que un tal poterea ancora salvar le patiente. Le autores propone que in casos equivoc un diagnose pragmatic de "tumor ossee a cellulas ronde" es formulate e que isto es sequite per intense irradiation. In supporto de lor argumento le autores reporta cinque casos, originalmente diagnosticate como sarcoma de Ewing e dominate con successo durante periodos de plus que cinque annos, le quales esseva identificate sub reevalutation como sarcomas a cellulas reticulari in tres patientes e como myelomas solitari in duo Le autores sublinea le importantia de biopsias adequate effectuate per exposition aperite, con preservation experte del specimens e examines de illos in le forma de sectiones paraffinate. Illes rejice irradiation como test diagnostic, tanto ante como in loco del biopsia. Es sublineate le importantia de irradiar le osso integre, includente le areas satis infra e supra le articulationes proximal, con un dose avantiate usque al limite del toleration histologic. Nos considera irradiation como tractamento de election in le majoritate del casos A vices illo servi a salvar un extremitate; a vices le tumor non es chirurgicamente accessibile

7

Fibrosarcoma

John C. Ivins, M.D.*

INTRODUCTION

It has been pointed out[6] that success in the treatment of any malignant disease depends to a great extent upon a knowledge of its origin and its course, and that the institution of adequate surgical measures based on this knowledge is just as important as the early recognition of the presence of the malignant process.

There is no general agreement as to either the origin or the course of the malignant tumor which we call *fibrosarcoma;* hence, it would be expected that the treatment of this tumor would be attended by uncertain success. Results as published in the literature seem to confirm this expectation.

Stout[20] has described a wide variety of clinical conditions which, in microscopic manifestations, are characterized by the proliferation of fibrocytes, accompanied by collagen and reticulin fibers, and "more or less resemble neoplasms." Included in this group which he calls *fibromatoses* are such tumors as fibromas, palmar and plantar fibromatosis or Dupuytren's contracture, desmoid lesions and others. He wrote that very few of these ever proceeded to the development of malignant processes, but, rather, that the latter were malignant from the start.

The foregoing, then, introduces the first difficulty, namely, that of separating the benign from the malignant lesions in the case of well-differentiated fibroblastic processes. In the case of poorly differentiated lesions, a second difficulty is encountered, and that is the trying task of separating fibrosarcomas from other malignant tumors of mesenchymal origin such as synoviomas, liposarcomas and rhabdomyosarcomas. Occasionally, it is difficult to establish a diagnosis of fibrosarcoma at all; in other cases, expert pathologists frequently will give widely divergent opinions when trying to predict the malignant potentialities of a given tumor which is agreed to be a fibrosarcoma.

Fibrosarcomas are not encountered often, and the accumulating knowledge about the origin and the course of the lesions is gained necessarily from the continuing study of diagnoses made and patients treated in the practices of large medical centers. The present commentary summarizes the experience with this tumor at the Mayo Clinic.

In 1934, Hargrave studied critically 152 cases in which fibrosarcoma of the soft tissues of the extremities was seen at the Mayo Clinic through 1930. Meyerding, Broders and Hargrave[4,15] later reported on these cases in 2 comprehensive papers that appeared in 1936 and 1939. Evidence was presented to show that the bulk of these fibroblastic tumors did not originate from peripheral nerve structures, and that a fairly accurate estimate of the degree of malignancy could be obtained by the histologic grading of these tumors. These and other conclusions were at variance with those in other major reports in the literature.

* Section of Orthopedic Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minnesota The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

An additional 78 cases in which this lesion was encountered during the years 1931 to 1940, inclusive, were analyzed in 1948.[13] These cases were reported in 1950,[14] and, among other things, the report showed that (1) the so-called 5-year survival rate for patients with fibrosarcoma, regardless of the pathologic grade of the tumor and of the treatment employed, was 38 per cent; (2) the survival rate associated with fibrosarcoma seemed to compare very favorably with the survival rate accompanying other major malignant processes; (3) fibrosarcomas of the soft tissues of the extremities could be grouped into 4 grades of relative malignancy based on the histopathologic structure; and that (4) this grouping into grades of relative malignancy had prognostic significance in that the survival period was inversely proportionate to the grade of malignancy.

My colleague Dr. C. R. Sullivan carried on this study to include all patients seen through 1946. These, added to the patients treated during the 1930's, gave a total of 107. Follow-up studies were carried out concerning all the patients,[12] and the results of these studies in 107 cases form the basis for this chapter.

DEFINITION

Fibrosarcoma may be defined as:

> a neoplasm of connective-tissue origin, the typical cell of which is the spindle cell, or fibroblast, which characteristically exhibits intercellular collagenous argyrophil fibers in varying amounts, and which is manifestly malignant as shown by its tendency to recur locally, by the property of metastasizing and by pleomorphic or anaplastic histologic appearance.

Numerous terms, apparently synonymous with fibrosarcoma, persist in the literature. Among these are *spindle cell sarcoma, round cell sarcoma, fibroblastic cell sarcoma* and *neurogenic sarcoma*

So far as the term *neurogenic sarcoma* is concerned, the trend in recent work appears to be away from its use. The title does not refer to an entity in any way distinct from fibrosarcoma and, according to Stout:[18]

> probably stems from the fact that the schwan- [illegible]
>
> malignant schwannoma may be mistaken for a fibrosarcoma

It is also significant that in the panel discussion on soft part tumors at the National Cancer Conference[19] in 1949, it was recorded that:

> after a recent review of several hundred tumors previously classified at the Memorial Hospital as neurogenic sarcoma, most of the tumors were removed from this category and the remainder are now designated schwannoma.

INCIDENCE

Fibrosarcoma is encountered relatively infrequently. Soule studied 500 consecutive soft tissue tumors of the extremities, excluding those of dermal origin, seen at the Mayo Clinic over a 3-year period, and found 77 which were malignant. Of these 77 malignant tumors, 31 (40.2%) were fibrosarcomas. Thus, fibrosarcoma comprised 6.2 per cent of the total number. It is the commonest malignant tumor of the soft tissues of the extremities, if those of dermal origin are excluded. On the basis of the literature,[13,14] it would seem to occur once in 2,000 to 4,000 cases.

ANATOMIC DISTRIBUTION

The majority of fibrosarcomas occur in the soft tissues of the trunk and the extremities. This is verified in all the major reports in the literature. Of the 107 tumors considered herein, 30 (28%) occurred in the upper limbs, while 77 (72%) were in the lower limbs. Of the 77 tumors encountered in the lower extremities, about two thirds (53, or 68.8%) were in the thigh or the region of the knee. Considering the group as a whole, about half were found in the lower part of the thigh or the region of the knee. This is in exact accord with the findings of Brindley and associates, who

reported 45 fibrosarcomas, 51 per cent of which were in the thigh. There seems to be no other explanation for the more common occurrence of this tumor in the thigh than that the thigh, which has a larger volume than other parts, contains more of the connective tissues from which fibrosarcomas arise.

GENERAL ETIOLOGIC FACTORS

Sex. The incidence of fibrosarcoma according to sex in this series was 63 tumors in men (58.9%) and 44 in women (41.1%). In other reports in the literature there are no striking differences in this incidence.

Age. Fibrosarcoma appears at any age. Hudson reported an intra-uterine fibrosarcoma of the foot, and many fibrosarcomas have been observed in the aged; however, persons chiefly affected are in the third, the fourth and the fifth decades. Our patients ranged from 12 to 76 years, with an average age of 45.4 years for the entire group. Brindley's patients averaged "approximately 45 years."

Trauma. Nearly a quarter (25) of our patients gave a history of trauma related in some way to the appearance of a fibrosarcoma. Trauma has been implicated many times as a possible etiologic factor in the production of certain tumors. The literature abounds with reports that purport to show trauma and tumor as cause and effect, and Ewing has stated that we cannot exclude trauma as a possible cause of many tumors. Two brief reports of cases will serve to illustrate the type of situation that continues to stimulate the thought that trauma does play some role in the production of these tumors.

Case 1. A 41-year-old man noted the onset of a rather hard lump on the right thigh a month after a rather severe local bruise. A Grade 2 fibrosarcoma was excised 3 months after the onset; a rapidly growing recurrent lesion was noted a month later; the patient died less than a year after the onset from pulmonary metastasis.

Case 2. A 48-year-old man had had a mass on the distal part of the right leg for 30 years. Seven years prior to his admission he had been kicked by a calf, after which the mass began to grow. Two excisions had been done for a Grade 3 fibrosarcoma; then amputation was carried out. The patient died of pulmonary metastasis 2 years after the first excision.

Heredity. Heredity seems to play no role as a predisposing factor in the development of fibrosarcoma. In this series, as in most others, about 20 per cent of the patients (20 of 107) gave a familial history of cancer. A survey of the general population probably would give a like figure.

SYMPTOMATOLOGIC ASPECTS

The patient with fibrosarcoma has little warning of his ominous disease. In all but 2 of our patients, the presence of a mass, or swelling, was the initial complaint. Only 2 of the 107 patients were aware of discomfort or pain preceding the appearance of a mass. Only 35 patients listed pain as a symptom associated with a palpable mass, and in only 3 cases was the pain severe.

The duration of symptoms varied from as little as a week to 30 years before the patients were seen at the Mayo Clinic. About two thirds of the patients (69, or 64.4%) presented with recurrent processes, 1 or more excisions having been done elsewhere.

There were no constant constitutional symptoms. Only 3 patients had lost weight. Results of routine laboratory examinations of the blood and the urine were equivocal in all cases. Routine roentgenologic examination of the involved part showed nothing characteristic; in 61 cases (57%) there was evidence of a soft tissue mass.

GROSS ANATOMIC FEATURES

Usually, fibrosarcomas originate in the subcutaneous tissues and fasciae of the extremities or from the fibrous intermuscular or intramuscular septa. Less commonly, they arise from the tendons and from the dermis. They may arise from the fibrous tissue investments, nerves and blood vessels, and at times from the fibrous layer of the perios-

teum. In the fresh specimen, or at the operating table, it is extremely difficult as a rule to determine exactly the tissue of origin. Smaller tumors may be encased within a single muscle. Definite origin in a nerve can rarely be demonstrated, but the large tumors commonly surround nerves and blood vessels.

The tumor usually is single, but at times it may be multicentric in origin, particularly in the case of recurrent tumors. On palpation the tumor is neither so hard as a carcinoma nor so soft as a lipoma. Characteristically, there is a firmness that is rather midway between these extremes; however, very cellular fibrosarcomas may give a soft, fluctuant sensation on occasion, whereas low grade very fibrous tumors may be nearly bony hard. The mass is rounded or lobulated, and, as a rule, it is rather sharply delimited from the surrounding normal tissue (Fig. 1).

In cut sections there is seen an apparent translucence that varies according to the amount of edema present. The soft tumors are very cellular, very vascular and homogeneous in texture. The hard tumors are fibrous, relatively nonvascular and fasciculated in appearance. The more rapidly growing tumors will show varying amounts of hemorrhage and scattered areas of necrosis. Occasionally, a truly myxomatous tumor will yield a gelatinous substance on cut section, but most fibrosarcomas described as *myxomatous* give negative results when stained for mucus, their myxomatous appearance being due to edema.

Encapsulation is said to be a characteristic feature of a large number of fibrosarcomas, but this encapsulation is more apparent than real. The tumor grows expansively. The peripheral portions grow apace with the more central portions. The lobulated feature is the result of perivascular growth. At the most peripheral portion of the mass the rapid expansion compresses the outermost

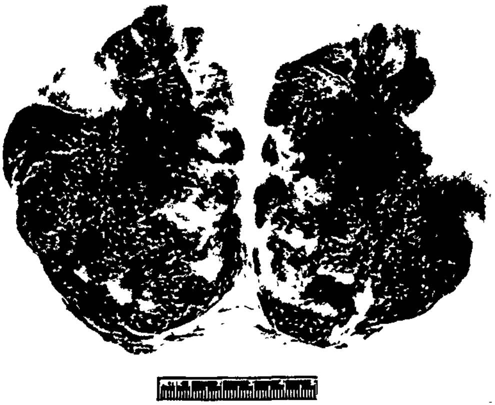


FIG. 1 Fibrosarcoma from the thigh, showing lobulated and circumscribed character of the gross specimen.

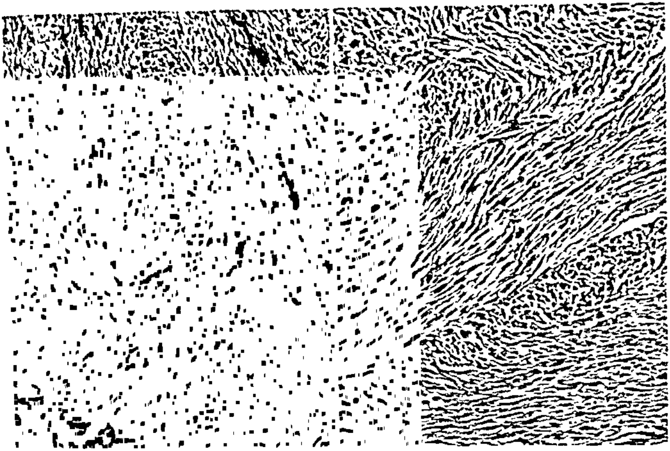


FIG. 2. (*Left*) Grade 1 fibrosarcoma; it is characterized by slender, small, well-differentiated fibroblasts arranged in strands (hematoxylin and eosin; × 140). (*Right*) Grade 2 fibrosarcoma; this is a more cellular tumor, with large cells featuring moderately hyperchromatic nuclei, but still definitely arranged in strands (hematoxylin and eosin; × 140).

tissue into a false capsule, from which fasciculi seem to run in interlobular strands, giving the cut section its typical fasciculated, multilobular appearance. This pseudocapsule, or *compression zone,* usually is invaded; or, more truthfully, it shows infiltrative growth at the periphery, by reason of which it is difficult to extirpate these tumors, and they recur commonly after excision. Extension into the lumens of blood vessels is common; hence the majority of these neoplasms metastasize rather early. There is nothing characteristic about the size of these tumors, and no relationship can be established among size of the tumor mass, pathologic grade, duration and end-result.

MICROSCOPIC ANATOMY

General. There is considerable variation in the microscopic structure of fibrosarcoma, just as there is in the gross appearance and the clinical behavior of the lesion. This neoplasm of mesodermal origin is composed of cells, the ultimate function of which is the production of fibrous connective tissue. Fibrosarcomas vary in structure within the limits of precursive tissue architecture and within the limits of adult connective tissue variations. They range from the very fibrous fibrosarcoma, closely akin to the benign fibroma, to the very cellular fibrosarcoma, which exhibits marked anaplasia and pleomorphism. Yet all of them have certain common features that distinguish fibrosarcomas as a definite group of tumors.

The type cell is the fibroblast, a cell form intermediate between the primitive, undifferentiated mesoblast from which it arises and its adult counterpart, the fibrocyte. These cells vary in their degree of differentiation from one tumor to another, but in any one given tumor there usually is a rather

reliable uniformity. In the more primitive tumors the cells are stellate, but typically they are fusiform or elongated and have been described by the familiar term *spindle cell.*

The chief function of these cells is fibrogenesis. They are separated by varying amounts of collagenous argyrophil fibrils arranged in strands. The amount of collagen present is an index of the degree of differentiation. Thus, the more adult tumors show more abundant amounts of collagen. The collagenous matrix generally is rather homogeneous, but at times it may be myxomatous in appearance. This may be due to edema subsequent to circulatory stasis, but at times it is due to the presence of mucoid substances in the matrix which give a positive reaction to thionine and mucicarmine stains recognized as specific for myxomatous tissue. Such myxomatous tissue may be associated with any neoplasm of mesodermal origin, but Bick has pointed out that all fibroblastic neoplasms need not pass through a myxomatous stage before becoming fibrous.

Grade 1 Fibrosarcoma (Fig. 2 *left*). Twenty-two of this entire series of tumors were classified as Grade 1, representing about a fifth of the group. These were fibrocellular tumors. None was so adult developmentally that the purely fibrous structure of benign fibroma was produced; likewise, none was so lacking in fibrogenesis that a purely cellular description would apply. As a group, these tumors exhibited abundant fibrogenesis, and the polarity of the cells and the fibers was additional evidence of a relatively adult tissue. The individual cellular elements showed little variation in size or shape. They were fusiform, not closely packed, and presented spindle-shaped nuclei which did not stain deeply. Mitotic figures, both normal and pathologic, were present in all the tumors, but in some such figures could be found in almost every field, whereas in others they were located only after the most diligent search. The tumors of this grade were not very vascular There was nothing characteristic about their location.

Grade 2 Fibrosarcoma (Fig. 2, *right*). Thirty-three of the tumors of this series were classified as Grade 2, representing about 31 per cent of the total. As a group, these tissues exhibited rather abundant production of fiber but noticeably less than was evident in the tumors classified as Grade 1. A few of the tissues were noted to contain tumor giant cells. Mitotic figures were present in increased numbers, a manifestation of increased cellular activity. The individual cellular elements, however, were not markedly different from those classified as Grade 1. Some of the tumors showed mild degrees of variation in size and in shape of the cells, but, in general, rather uniform spindle cells predominated. The whole group was more vascular than the Grade 1 tumors.

Grade 3 Fibrosarcoma (Fig. 3, *left*). Thirty-four of the tumors of this series were classified as Grade 3, again representing about 31 per cent of the total. Most were fibrocellular in their general character; however, even though there was evident production of fiber, these tissues all were very much more cellular than those classified as Grades 1 and 2. As a rule, the individual cellular elements tended to be smaller and more closely packed and more darkly stained. Variation in cellular size and shape was greater here than in the case of the lower grade tumors; in some of them there were spindle-shaped cells, but generally the cells tended to be more plump or rounded in appearance. Rather striking was the increased number of mitotic figures to be found in the tissues of this group. In almost all the tumors, at least 1 mitotic figure could be found in most oil-immersion fields. All manner of so-called abnormal mitotic figures could be found with some search. Also, the increased number of tumor giant cells to be found was a striking feature. Polarity was not a prominent feature, both because of the reduced fibrogenesis and the change in the general shape of the cells. Scattered areas of hemorrhage and hyaline necrosis were seen much more frequently in these tumors than in

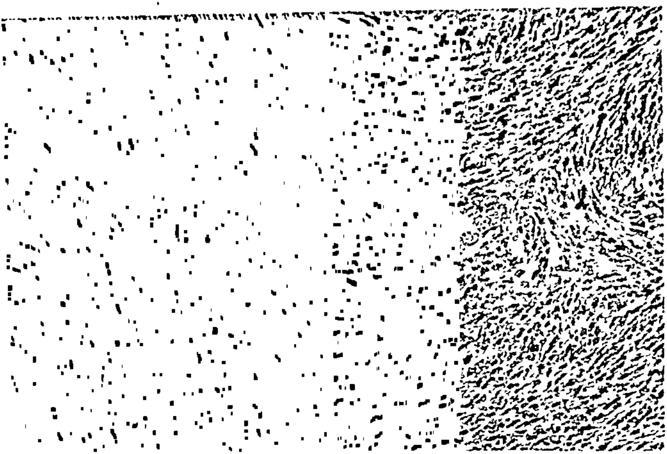


FIG. 3. (*Left*) Grade 3 fibrosarcoma; note the lack of orderly arrangement, increased cellularity and mitotic figures (hematoxylin and eosin; × 140). (*Right*) Grade 4 fibrosarcoma; a highly anaplastic cellular tumor showing pathologic mitotic figures and decreased fibrogenesis (hematoxylin and eosin; × 140).

those of the lower grades, and, as a group, they were rather well supplied with thin-walled newly formed blood vessels.

Grade 4 Fibrosarcoma (Fig. 3, *right*). The remaining 18 tumors in this group of 107 were classified as Grade 4. The tissues of these lesions all were very cellular as compared with those of tumors of lower grades; however, in all there was some degree of fibrogenesis, which usually was present only in scattered areas rather than being uniformly present, so that as regards their general character the tumors were classed as *fibrocellular*, as were the tumors of the other grades. Characteristic of the individual cellular elements seen in these Grade 4 tumors was the very marked pleomorphism. In some, there were areas in which rather plump spindle cells could be found; generally, however, the cellular elements were of all sizes and shapes—from small round cells with very little cytoplasm and a dark dense nucleus to very large tumor giant cells having a granular cytoplasm, 1 to 5 or more nuclei, and small dark nucleoli. These tumor giant cells were conspicuous features, even on examination with the low power lens, with which large numbers of them could be seen in most of the tissues of this group. Mitotic figures, both normal and pathologic, were present in great numbers in these tumors, the section of average thickness usually containing at least 1 in every oil-immersion field.

The foregoing features combined to give the tissues of this group a very disordered, *wild* or *dirty* appearance in contrast with the more or less orderly appearance of most of the tumors in the lower grades. Newly formed vessels were much in evidence, but the frequency of the patchy necrosis seen in these tissues testified to the rapidity with which they grew. Another manifestation of

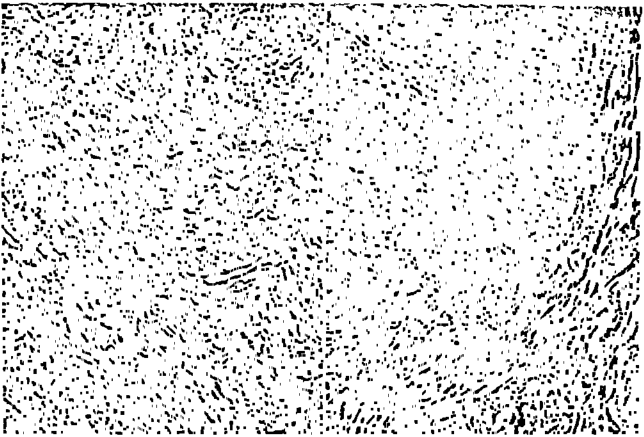


FIG. 4. (*Left*) Fibroma; well-differentiated fibrocytes producing dense, wavy collagen (hematoxylin and eosin; × 140). (*Right*) Palmar fibromatosis; tissue from hand showing nodular fibroblastic proliferation arising in palmar fascia (hematoxylin and eosin; × 140).

rapid growth was the difference in the character of the *pseudocapsule* seen in the Grade 4 tumors. Whereas in the tumors of lower grade this compression zone was composed of tightly packed spindle cells and fibers, in the tumors of Grade 4 there was only the suggestion of compression at the periphery, and the general character of the cells in the peripheral zone was little changed from that of the cells in the center of the neoplastic mass.

DIFFERENTIAL DIAGNOSIS

The microscopic diagnosis of fibrosarcoma usually is evident from the characteristic structure However, there are several neoplasms, both benign and malignant, which at times serve to make interpretation of the histologic appearance difficult.

Of the benign tumors (or tumorlike conditions), the following are the most important from the standpoint of microscopic differential diagnosis:

Fibroma (Fig. 4, *left*). Pure fibroma is not a common tumor, but benign histologic variants are found rather frequently to be widely distributed throughout the body. In the extremities they occur at subcutaneous sites and in the intermuscular and the fascial planes; in these situations they cannot be distinguished clinically from fibrosarcoma. Microscopically, the thick bundles of dense collagenous connective tissue, with interspersed small flattened and elongated nuclei, make the diagnosis clear. However, it is very difficult to distinguish the more cellular fibromas from Grade 1 fibrosarcoma, so that every cellular fibroma should be looked upon with suspicion. Histologically, there is no certain way to make this important

distinction, and, as Soule has pointed out, there appears to be a *twilight zone* in which benign and malignant forms blend. Clinically, the issue is equally confused; except for the presence of a mass, neither tumor produces symptoms. Even the most benign fibroma must have grown at some time in its existence; on the other hand, tissues that manifestly are malignant have been known to lead a static existence of many months or years.

Fibromatosis (Fig. 4, *right*). Nodular fibrosis of the palmar or plantar fascia, usually called *Dupuytren's contracture*, is very common. There are several features about this condition that make distinction from fibrosarcoma most difficult at times. First, in the excised fascia there is histologic evidence of an active proliferation of fibroblasts without other signs of inflammation. Next, the course of the contracture may be rapid, or it may be very slow, and there is some tendency for the condition to recur; this is interpreted by some as not representing a true recurrence but, rather, a spread of the condition from fascia *in situ* that was not excised primarily. One brief report will illustrate the above points:

Case 3. A 13-year-old girl was admitted for examination because of albuminuria and puffy eyes. During the course of the examination a small mass was noted on the plantar surface of the left foot. There were no symptoms, and nothing was known of the duration of the mass. The mass was excised, and the tissue was reported as *dense fibroma.* Ten months later a recurrence was excised, and the diagnosis was *dense fibroma with areas of Grade 1 fibrosarcoma.* One year later a second recurrence was excised, and the histologic diagnosis was the same Nineteen months later a third recurrence was excised, and the examination showed *inflammatory fibrous tissue.* No further trouble was encountered, and the patient remained well. A review of all the tissue in this case showed typical nodular fibroblastic proliferation, and the diagnosis was revised accordingly.

Desmoid Tumor (Fig. 5). Desmoid tumors are benign fibrous neoplasms arising from the musculo-aponeurotic structures



FIG. 5. Extra-abdominal desmoid tumor; infiltrative fibroblastic growth surrounding degenerating striated muscle (hematoxylin and eosin; × 140).

throughout the body, which have the peculiar characteristic locally of invading and engulfing the adjacent striped muscle fibers. Therefore, morphologically they have the local malignant property of invasion, but histologically they are benign tumors. At times it may be most difficult to distinguish this tumor from fibrosarcoma. A desmoid is not a common tumor. The vast majority of them occur in the substance of the rectus abdominis muscle of parous women; however, some 10 per cent of them occur in extra-abdominal sites. The type cell is the fibroblast, which in the more active tumors may be embryonic, with large nuclei, prominent nucleoli and rather abundant cytoplasm. Mitotic figures can be found without difficulty. Most characteristic are the so-called giant cells, which consist of disintegrating fragments of striated muscle that at times show proliferating nuclei. These are the mul-

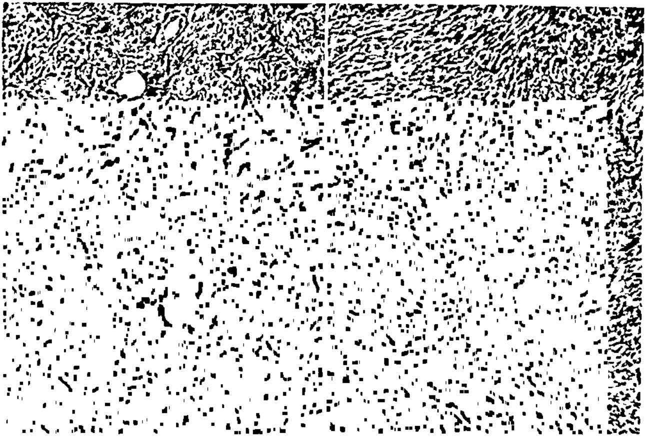


FIG. 6 Synovial sarcoma. (*a*) Typical bimorphic cells resembling metastatic adenocarcinoma (hematoxylin and eosin, × 140). (*b*) Spindle-cell area suggestive of fibrosarcoma (hematoxylin and eosin, × 140).

tinucleated plasmodial masses of Boyd. Deposits of hemosiderin here and there throughout the tissue may be surrounded by nests of inflammatory cells

At times it may be difficult to differentiate 3 malignant tumors from fibrosarcoma:

Synovial Sarcomas:

> Synovial sarcoma may be defined as a malignant mesenchymal tumor that is composed of bimorphic cells and that usually is found arising near, but not necessarily from, the lining membranes of joint capsules, bursal sacs and tendon sheaths.[7]

They are said to account for 10 per cent of all primary malignant tumors of the soft parts of the extremities and are regarded as the commonest sarcoma of the hands and the feet. Microscopically there are 2 basic cellular types: first, a fibroblastlike cell capable of producing collagen and giving a fibrosarcomatous matrix to support the second cell type; and second, endothelial cells, which vary from flattened spindle elements frequently lining clefts to cuboidal cells that arrange themselves about glandlike spaces. Tufting of these endothelial cells and the production of mucus may simulate a mucous adenocarcinoma (Fig. 6, *left*). In the typical tumor the mixture of carcinomalike and sarcomalike elements is fairly easily recognized as a synovial sarcoma; however, when the fibrosarcomatous matrix predominates, distinction from fibrosarcoma may be most difficult (Fig. 6, *right*).

Liposarcoma. Liposarcomas are rare tumors, representing only 3 per cent of all primary lipomatous tumors of the extremities;[17] however, most of them occur in the lower extremities, where fibrosarcomas are commonly found, and in which situation they cannot be distinguished clinically from other soft tissue tumors. Microscopically there is a wide variation in appearance. The malignant lipoblasts, which serve to identify the

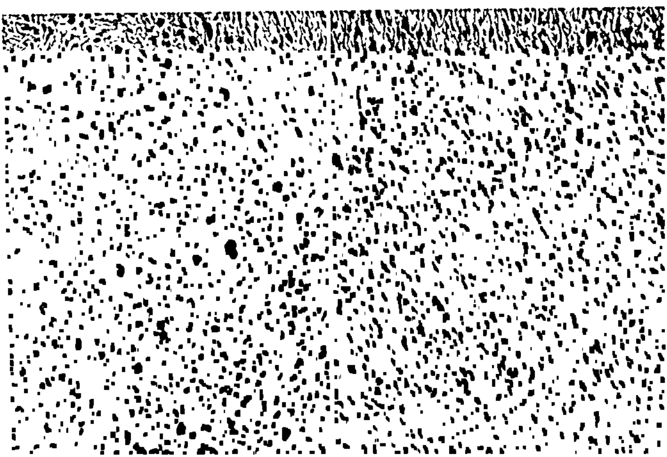


FIG. 7. Liposarcoma. (*a*) Malignant lipoblasts, showing foamy fat-containing cytoplasm (hematoxylin and eosin; × 140). (*b*) Spindle-cell element resembling fibrosarcoma (hematoxylin and eosin; × 140).

neoplasm, are pleomorphic cells appearing as signet cells, spindle cells and large bizarre giant cells with foamy cytoplasm (Fig. 7, *left*). Soule has pointed out that myxomatous areas frequently are encountered, particularly in the tumors of lower grades of malignancy, and that the presence of spindle cells, giant cells and a myxomatous appearance may cause the lesion to resemble fibrosarcoma very closely (Fig. 7, *right*).

Rhabdomyosarcoma. A definite pathologic entity are tumors arising from muscle tissue. Whether these malignant tumors arise from existing adult muscle fibers or from immature skeletal muscle elements is still in dispute. Although rare, they may be found in any age group, the peak of incidence being in the fifth decade. Clinically, they cannot be distinguished from other soft tissue masses. The primary cells of the tumor are spindle cells which, when special staining technics are used, may be shown to contain fibrils sometimes exhibiting beading or striations. In addition to these basic spindle cells, there is present a mixture of tumor giant cells, round cells, strap-shaped cells with 2 or more nuclei in tandem, or racket-shaped cells with a single nucleus at 1 expanded rounded end. It may be very difficult to distinguish such a rhabdomyosarcoma from a Grade 4 fibrosarcoma when there also is marked pleomorphism with many giant cells (Fig. 8).

TREATMENT AND RESULTS

Generally it is agreed that the treatment of fibrosarcoma is unsatisfactory. Just how unsatisfactory such treatment is it is difficult to judge on the basis of reports in the literature; and this difficulty is due to several factors. First, the passing years have produced changes in diagnostic concepts, which means that the older reports have very limited value. For example, in study of the



FIG. 6. Synovial sarcoma. (*a*) Typical bimorphic cells resembling metastatic adenocarcinoma (hematoxylin and eosin; × 140). (*b*) Spindle-cell area suggestive of fibrosarcoma (hematoxylin and eosin; × 140).

tinucleated plasmodial masses of Boyd. Deposits of hemosiderin here and there throughout the tissue may be surrounded by nests of inflammatory cells.

At times it may be difficult to differentiate 3 *malignant tumors from fibrosarcoma:*

Synovial Sarcomas:

Synovial sarcoma may be defined as a malignant mesenchymal tumor that is composed of bimorphic cells and that usually is found arising near, but not necessarily from, the lining membranes of joint capsules, bursal sacs and tendon sheaths.[7]

They are said to account for 10 per cent of all primary malignant tumors of the soft parts of the extremities and are regarded as the commonest sarcoma of the hands and the feet. Microscopically there are 2 basic cellular types: first, a fibroblastlike cell capable of producing collagen and giving a fibrosarcomatous matrix to support the second cell type; and second, endothelial cells, which vary from flattened spindle elements frequently lining clefts to cuboidal cells that arrange themselves about glandlike spaces. Tufting of these endothelial cells and the production of mucus may simulate a mucous adenocarcinoma (Fig. 6, *left*). In the typical tumor the mixture of carcinomalike and sarcomalike elements is fairly easily recognized as a synovial sarcoma; however, when the fibrosarcomatous matrix predominates, distinction from fibrosarcoma may be most difficult (Fig. 6, *right*).

Liposarcoma. Liposarcomas are rare tumors, representing only 3 per cent of all primary lipomatous tumors of the extremities;[17] however, most of them occur in the lower extremities, where fibrosarcomas are commonly found, and in which situation they cannot be distinguished clinically from other soft tissue tumors. Microscopically there is a wide variation in appearance. The malignant lipoblasts, which serve to identify the

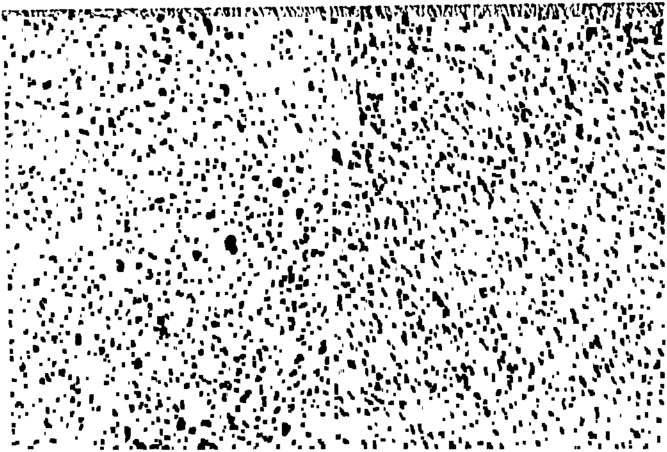


FIG. 7. Liposarcoma. (*a*) Malignant lipoblasts, showing foamy fat-containing cytoplasm (hematoxylin and eosin; × 140). (*b*) Spindle-cell element resembling fibrosarcoma (hematoxylin and eosin; × 140).

neoplasm, are pleomorphic cells appearing as signet cells, spindle cells and large bizarre giant cells with foamy cytoplasm (Fig. 7, *left*). Soule has pointed out that myxomatous areas frequently are encountered, particularly in the tumors of lower grades of malignancy, and that the presence of spindle cells, giant cells and a myxomatous appearance may cause the lesion to resemble fibrosarcoma very closely (Fig. 7, *right*).

Rhabdomyosarcoma. A definite pathologic entity are tumors arising from muscle tissue. Whether these malignant tumors arise from existing adult muscle fibers or from immature skeletal muscle elements is still in dispute. Although rare, they may be found in any age group, the peak of incidence being in the fifth decade. Clinically, they cannot be distinguished from other soft tissue masses. The primary cells of the tumor are spindle cells which, when special staining technics are used, may be shown to contain fibrils sometimes exhibiting beading or striations. In addition to these basic spindle cells, there is present a mixture of tumor giant cells, round cells, strap-shaped cells with 2 or more nuclei in tandem, or racket-shaped cells with a single nucleus at 1 expanded rounded end. It may be very difficult to distinguish such a rhabdomyosarcoma from a Grade 4 fibrosarcoma when there also is marked pleomorphism with many giant cells (Fig. 8).

TREATMENT AND RESULTS

Generally it is agreed that the treatment of fibrosarcoma is unsatisfactory. Just how unsatisfactory such treatment is it is difficult to judge on the basis of reports in the literature; and this difficulty is due to several factors. First, the passing years have produced changes in diagnostic concepts, which means that the older reports have very limited value. For example, in study of the

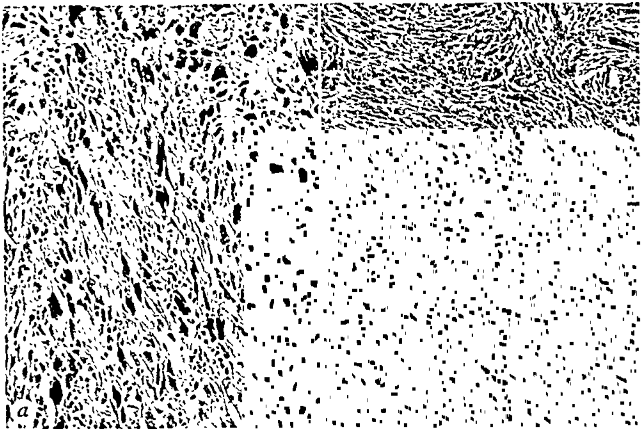


FIG. 8. Rhabdomyosarcoma. (*a*) Giant rhabdomyoblasts, featuring pink-staining granular cytoplasm (hematoxylin and eosin; × 140). (*b*) Small spindle-cell element devoid of granular cytoplasm and resembling fibrosarcoma (hematoxylin and eosin; × 140).

material used as the basis of this report, 26 cases were discarded because the original diagnosis was found to be incorrect, an evidence of the increasing diagnostic acumen of our pathologists.

Second, many of the reports list as *cured* those patients known to have been alive as short a time as 3 years after surgical treatment when, as a matter of fact, the present study has shown that local recurrence or metastasis may appear many years after primary treatment

Third, lack of clear definition of terms demands that evaluation of any form of treatment be done with the greatest restraint, since what is considered to be wide, radical or en bloc excision by one surgeon may be considered conservative by another In the present series of cases, in which so many patients underwent one or several conservative surgical procedures done elsewhere, with or without adjunctive radium or roentgen therapy, lead therapy or the use of Coley's toxins, it is impossible to produce figures which prove that any one particular treatment is superior.

At present the generally accepted treatment of fibrosarcoma of the extremities appears to be en bloc excision of low grade lesions, amputation of less well-differentiated neoplasms or recurrences, and roentgen therapy for surgically inaccessible tumors. However, the recent reports of Stout[18] and Cade indicate that recurrences have afflicted 60 per cent of patients, and that death from tumor has come to 23 and 50 per cent, respectively. Pack reported that more than 40 per cent of patients died of their disease in less than 5 years. Obviously, the current management is none too good

The table on page 79 indicates the outcome for 94 of the 107 patients in this re-

SURVIVAL AMONG 107 PATIENTS WHO HAD FIBROSARCOMA OF THE SOFT TISSUES OF THE EXTREMITIES

Lesion, Grade	Patients, Number	Condition Followed	Died of Tumor	Died,* Cause Unknown	Living, with Disease	Alive and Well (Years After Last Surgery)				
						0-4	5-9	10-14	15-19	20+
1	22	18	5 (27.7%)	3	0	3	4	1	2	0
2	33	26	14 (53.8%)	4	0	3	0	1	4	0
3	34	33	21 (63.6%)	1	1	1	3	4	2	0
4	18	17	12 (70.5%)	3	0	0	0	1	0	1
Total	107	94	52 (55.3%)	11	1	7	7	7	8	1

* Cause of death either was unknown or was disease other than tumor.

port. Of the 94 patients whose condition was followed, 52 (55.3%) died of fibrosarcoma. Of those who died, the average duration of life after the onset of disease was 6.6 years. Patients with histologically well-differentiated tumors lived longer than those who had poorly differentiated lesions. For patients who had Grade 1, 2, 3 or 4 tumors, the average duration of life was 11.2, 8.0, 6.1 and 4.1 years, respectively. Thus it becomes apparent that histopathologic grading of the degree of malignancy is of prognostic significance when the death rate from the tumor is computed. This is shown in Column 4 of the table.

No unequivocal statement can be made concerning the choice of proper treatment of fibrosarcoma of the soft tissues of the extremities, particularly in the matter of primary amputation as opposed to local excision of the initial lesion. Only 8 of the 107 patients were treated primarily by amputation of the diseased extremity, and of these only 1 was living and well 14 years after operation. No follow-up data are available concerning 2 patients, and 5 are known to have died of tumor. This, however, is not a fair appraisal of the efficacy of primary amputation. Speaking of his own failures after amputation, Cade wrote:

> It is obvious that those submitted to amputation were more advanced than those where wide local excision appeared to offer a reasonable chance.

Friedman, commenting on this, said:

> . . . the relatively innocent appearance and behavior of many primary tumors generally misleads the surgeon to undertake conservative procedures which are inadequate. As a result the five year cure is low, 20 per cent or less, instead of high, as would theoretically be expected of a tumor which is generally slow-growing, of low degree of malignancy, usually accessible and detectable moderately early.

It is evident that, on the whole, local excision of fibrosarcoma is inadequate. It is equally evident that the price of a high rate of cure would be the needless sacrifice of some limbs were primary amputation performed in each instance. It remains a question as to which course is more worth while.

SUMMARY

Experience with 107 patients who had fibrosarcoma of the soft tissues of the extremities seen at the Mayo Clinic during a 15-year period has been reviewed. Careful study of this group of patients has shown the following.

1. Present methods of treatment leave much to be desired. Well over half of this group of patients died of their disease, and it appears that this situation might have been remedied, at least in part, by more radical primary treatment.

2. Histopathologic grading of fibrosarcomas into 4 relative grades of malignancy has prognostic significance in that, in this series, patients with histologically well-differentiated tumors lived longer than those who had poorly differentiated lesions.

REFERENCES

1. Bick, E. M.: Fibrosarcoma of the extremities, Ann. Surg. 101:759, 1935.
2. Boyd, William: Surgical Pathology, ed. 6, Philadelphia, Saunders, 1947.
3. Brindley, H. H., Phillips, Charles, and Fernandez, J. N.: Fibrosarcoma of the extremities: review of forty-five cases, J. Bone & Joint Surg. 37-A:602, 1955.
4. Broders, A. C., Hargrave, Robert, and Meyerding, H. W.: Pathological features of soft tissue fibrosarcoma with special reference to the grading of its malignancy, Surg., Gynec. & Obst. 69:267, 1939.
5. Cade, Stanford: Soft tissue tumours: their natural history and treatment, Proc. Roy. Soc. Med. 44:19, 1951.
6. Carroll, W. W.: Principles involved in surgical therapy of "encapsulated" fibrosarcoma of soft tissues, Surg., Gynec. & Obst. 84:703, 1947.
7. Dockerty, M. B.: Synovial sarcomas. *In* American Academy of Orthopaedic Surgeons: Instructional Course Lectures, vol 11, p. 45, Ann Arbor, Michigan, J. W. Edwards, 1954.
8. Ewing, James. Modern attitude toward traumatic cancer, Arch. Path. 19:690, 1935.
9. Friedman, Milton· The treatment of connective tissue fibrosarcoma with surgery and radium, Bull Hosp. Joint Dis. 4:66, 1943.
10. Hargrave, R. L.: Soft Tissue Fibrosarcoma of the Extremities. Thesis, Graduate School, University of Minnesota, 1934.
11. Hudson, C. C.: Intra-uterine fibrosarcoma of the foot: a case report, Am J. Cancer 26:568, 1936.
12. Ivins, J. C.. Fibrosarcoma of the soft tissues of the extremities *In* American Academy of Orthopaedic Surgeons: Instructional Course Lectures, vol 11, p 18, Ann Arbor, Michigan, J. W Edwards, 1954.
13. ——— Fibrosarcoma of the Soft Tissues of the Extremities. Thesis, Graduate School, University of Minnesota, 1948.
14. Ivins, J. C., Dockerty, M. B , and Ghormley, R K.. Fibrosarcoma of the soft tissues of the extremities: a review of seventy-eight cases, Surgery 28:495, 1950.
15. Meyerding, H. W., Broders, A. C., and Hargrave, R. L.: Clinical aspects of fibrosarcoma of the soft tissues of the extremities, Surg., Gynec. & Obst. 62:1010, 1936.
16. Pack, G. T.: End results in the treatment of sarcomata of the soft somatic tissues, J. Bone & Joint Surg. 36-A:241, 1954.
17. Soule, E. H.: Primary soft-tissue tumors of the extremities: classification: histogenesis and incidence. *In* American Academy of Orthopaedic Surgeons: Instructional Course Lectures, vol. 11, p. 3, Ann Arbor, Michigan, J. W. Edwards, 1954.
18. Stout, A. P.: Fibrosarcoma: the malignant tumor of fibroblasts, Cancer 1:30, 1948.
19. ———: (Moderator) Panel on soft part tumors, Proceedings of the First National Cancer Conference, American Cancer Society and National Cancer Institute of the U. S. Public Health Service, p. 206, Washington and New York, Federal Security Agency, 1949.
20. ———: Tumor of the Soft Tissues. Sec. 2, [illegible] Forces In- [illegible]
21. [illegible] the Soft Tissues of the Extremities. Thesis, Graduate School, University of Minnesota, 1954.

Fibrosarcoma

Summario in Interlingua

On ha signalate que le successo in le tractamento de omne maligne morbo depende a un alte grado de nostre cognoscentia de su origine e su curso e que le execution de adequate mesuras chirurgic super le base de ille cognoscentia es tanto importante como le prompte recognition del presentia de un processo maligne. Il non ha consenso del autoritates relative a o le origine o le curso del tumor maligne que nos appella fibrosarcoma. Ergo on debe expectar que le tractamento de iste tumor es characterisate per incertitude de successo. Le resultatos publicate in le litteratura pare confirmar iste expectation. Le prime difficultate es separar le benigne ab le maligne lesiones in le caso de ben-differentiate processos fibroblastic, como per exemplo fibromas, retraction del

aponeurose palmar, lesiones desmoide, etc. In le caso de mal-differentiate lesiones, le secunde difficultate se presenta. Illo es separar fibrosarcomas ab altere maligne tumores de origine mesenchymal, como per exemplo synoviomas, liposarcomas, e rhabdomyosarcomas. Proque fibrosarcomas non se incontra frequentemente, nostre cognoscentias cumulative del origine e del curso de iste lesiones resulta necessarimente ab le continue studio del diagnoses establite e del patientes tractate in le practica del grande centros medical. In le presente reporto le experientias del Clinica Mayo in iste campo es summarisate. Es studiate in detalio un total de 107 fibrosarcomas del histos molle del extremitates. Illos es gruppate secundo lor grado relative de malignitate. Gruppo I representa le grado infime de malignitate e gruppo IV le grado supreme. Es presentate detalios del grossier e microscopic anatomia, e importante aspectos del diagnose differential es considerate in detalio. Ex le gruppo de 107 patientes nos poteva traciar 94 pro studios de superviventia. Cinquanta-duo (55,3 pro cento) moriva de fibrosarcoma. Inter le mortes le superviventia median post le declaration del morbo esseva 6,6 annos. Patientes con histologicamente ben-differentiate tumores viveva plus longe que patientes qui habeva mal-differentiate lesiones. Pro le patientes con tumores de grado I, II, III, e IV, le superviventia median esseva 11,2, 8,0, 6,1, e 4,1 annos respectivemente. Assi il deveni apparente que le graduation histopathologic del malignitate es de signification prognostic quando le mortalitate causate per le tumor es computate. Il non es possibile formular un inequivoc judicamento in re le tractamento de election in casos de fibrosarcoma del histos molle del extremitates, specialmente relative al question del amputation primari per contrasto al excision local del lesiones initial. Solmente 8 del 107 patientes habeva essite tractate primarimente per amputation del extremitate afficite, e de istes solmente 1 esseva vive e ben 14 annos post le operation. Duo alteres non poteva esser traciate, e 5 esseva cognoscitemente morte ab le tumor. Tamen, iste datos non representa un juste evalutation del efficacia de amputation primari. Il pare evidente super le base de nostre studios que excision local de fibrosarcoma es generalmente inadequate. Il es equalmente evidente que le costo de un alte procentage de curationes obtenite per amputationes primari executate in omne caso esserea le sacrificio innecessari de un considerabile numero de extremitates. Le qual del duo cursos es plus recommendabile remane un question indecise. Il non pare que le prognose es influentiate per le tractamento adjunctive con radios Roentgen, toxina de Coley, o altere mesuras.

2. Histopathologic grading of fibrosarcomas into 4 relative grades of malignancy has prognostic significance in that, in this series, patients with histologically well-differentiated tumors lived longer than those who had poorly differentiated lesions.

REFERENCES

1. Bick, E. M.: Fibrosarcoma of the extremities, Ann Surg 101:759, 1935.
2. Boyd, William: Surgical Pathology, ed. 6, Philadelphia, Saunders, 1947.
3. Brindley, H. H., Phillips, Charles, and Fernandez, J. N.: Fibrosarcoma of the extremities: review of forty-five cases, J. Bone & Joint Surg. 37-A:602, 1955.
4. Broders, A. C., Hargrave, Robert, and Meyerding, H. W.: Pathological features of soft tissue fibrosarcoma with special reference to the grading of its malignancy, Surg., Gynec. & Obst. 69:267, 1939.
5. Cade, Stanford: Soft tissue tumours: their natural history and treatment, Proc. Roy. Soc. Med. 44:19, 1951.
6. Carroll, W. W.: Principles involved in surgical therapy of "encapsulated" fibrosarcoma of soft tissues, Surg., Gynec. & Obst. 84:703, 1947.
7. Dockerty, M. B.: Synovial sarcomas *In* American Academy of Orthopaedic Surgeons· Instructional Course Lectures, vol. 11, p. 45, Ann Arbor, Michigan, J. W Edwards, 1954.
8. Ewing, James: Modern attitude toward traumatic cancer, Arch Path. 19:690, 1935.
9. Friedman, Milton. The treatment of connective tissue fibrosarcoma with surgery and radium, Bull. Hosp. Joint Dis. 4:66, 1943.
10. Hargrave, R. L. Soft Tissue Fibrosarcoma of the Extremities. Thesis, Graduate School, University of Minnesota, 1934.
11. Hudson, C. C. *Intra-uterine fibrosarcoma* of the foot: a case report, Am J. Cancer 26:568, 1936
12. Ivins, J. C.. Fibrosarcoma of the soft tissues of the extremities. *In* American Academy of Orthopaedic Surgeons· Instructional Course Lectures, vol. 11, p. 18, Ann Arbor, Michigan, J W. Edwards, 1954
13. ———. Fibrosarcoma of the Soft Tissues of the Extremities. Thesis, Graduate School, University of Minnesota, 1948
14. Ivins, J. C., Dockerty, M. B., and Ghormley, R. K : Fibrosarcoma of the soft tissues of the extremities: a review of seventy-eight cases, Surgery 28:495, 1950.
15. Meyerding, H. W., Broders, A. C., and Hargrave, R. L.: Clinical aspects of fibrosarcoma of the soft tissues of the extremities, Surg., Gynec. & Obst. 62:1010, 1936.
16. Pack, G. T.: End results in the treatment of sarcomata of the soft somatic tissues, J. Bone & Joint Surg. 36-A:241, 1954.
17. Soule, E. H.: Primary soft-tissue tumors of the extremities: classification: histogenesis and incidence. *In* American Academy of Orthopaedic Surgeons: Instructional Course Lectures, vol. 11, p. 3, Ann Arbor, Michigan, J. W. Edwards, 1954.
18. Stout, A. P.: Fibrosarcoma: the malignant tumor of fibroblasts, Cancer 1:30, 1948.
19. ———: (Moderator) Panel on soft part tumors, Proceedings of the First National Cancer Conference, American Cancer Society and National Cancer Institute of the U. S. Public Health Service, p. 206, Washington and New York, Federal Security Agency, 1949.
20. ———: Tumor of the Soft Tissues. Sec. 2, [illegible] Forces In- [illegible]
21. [illegible] the Soft [illegible] Graduate School, University of Minnesota, 1954.

Fibrosarcoma

Summario in Interlingua

On ha signalate que le successo in le tractamento de omne maligne morbo depende a un alte grado de nostre cognoscentia de su origine e su curso e que le execution de adequate mesuras chirurgic super le base de ille cognoscentia es tanto importante como *le prompte recognition del presentia de* un processo maligne. Il non ha consenso del autoritates relative a o le origine o le curso del tumor maligne que nos appella fibrosarcoma. Ergo on debe expectar que le tractamento de iste tumor es characterisate per incertitude de successo. Le resultatos publicate in le litteratura pare confirmar iste expectation. Le prime difficultate es separar le benigne ab le maligne lesiones in le caso de ben-differentiate processos fibroblastic, como per exemplo fibromas, retraction del

aponeurose palmar, lesiones desmoide, etc. In le caso de mal-differentiate lesiones, le secunde difficultate se presenta. Illo es separar fibrosarcomas ab altere maligne tumores de origine mesenchymal, como per exemplo synoviomas, liposarcomas, e rhabdomyosarcomas. Proque fibrosarcomas non se incontra frequentemente, nostre cognoscentias cumulative del origine e del curso de iste lesiones resulta necessarimente ab le continue studio del diagnoses establite e del patientes tractate in le practica del grande centros medical. In le presente reporto le experientias del Clinica Mayo in iste campo es summarisate. Es studiate in detalio un total de 107 fibrosarcomas del histos molle del extremitates. Illos es gruppate secundo lor grado relative de malignitate. Gruppo I representa le grado infime de malignitate e gruppo IV le grado supreme. Es presentate detalios del grossier e microscopic anatomia, e importante aspectos del diagnose differential es considerate in detalio. Ex le gruppo de 107 patientes nos poteva traciar 94 pro studios de *superviventia*. *Cinquanta*-*duo* (55,3 pro cento) moriva de fibrosarcoma. Inter le mortes le superviventia median post le declaration del morbo esseva 6,6 annos. Patientes con histologicamente ben-differentiate tumores viveva plus longe que patientes qui habeva mal-differentiate lesiones. Pro le patientes con tumores de grado I, II, III, e IV, le superviventia median esseva 11,2, 8,0, 6,1, e 4,1 annos respectivemente. Assi il deveni apparente que le graduation histopathologic del malignitate es de signification prognostic quando le mortalitate causate per le tumor es computate. Il non es possibile formular un inequivoc judicamento in re le tractamento de election in casos de fibrosarcoma del histos molle del extremitates, specialmente relative al question del amputation primari per contrasto al excision local del lesiones initial. Solmente 8 del 107 patientes habeva essite tractate primarimente per amputation del extremitate afficite, e de istes solmente 1 esseva vive e ben 14 annos post le operation. Duo alteres non poteva esser traciate, e 5 esseva cognoscitemente morte ab le tumor. Tamen, iste datos non representa un juste evalutation del efficacia de amputation primari. Il pare evidente super le base de nostre studios que excision local de fibrosarcoma es generalmente inadequate. Il es equalmente evidente que le costo de un alte procentage de curationes *obtenite per amputationes primari* executate in omne caso esserea le sacrificio innecessari de un considerabile numero de extremitates. Le qual del duo cursos es plus recommendabile remane un question indecise. Il non pare que le prognose es influentiate per le tractamento adjunctive con radios Roentgen, toxina de Coley, o altere mesuras.

8

Radiologic Aspects of Giant-Cell Tumor of Bone

ROBERT S. MACINTYRE, M.D., HOWARD B. LATOURETTE, M.D., AND FRED J. HODGES, M.D.*

To develop an accurate concept of any bone lesion requires that all its characteristics be recognized and correlated. Full understanding of the lesion usually designated as *giant-cell tumor of bone* is dependent upon familiarity with its roentgenologic appearance and its gross and histologic structure, recognition of its variable pattern of growth and biologic behavior, and experience with its response to various forms of treatment Such understanding now seems to have been developed to a point of considerable accuracy.

In reaching its present form, the concept of giant-cell tumor of bone has progressed through a number of stages reflecting growing knowledge of osseous physiology and pathology, and increasing clinical experience with the entity. Originally it was considered to be definitely malignant and, therefore, was treated by amputation of the involved extremity. Gradually it became apparent that in many instances the lesions included in this category were entirely benign in nature, and that healing might be expected following any one of a variety of treatment methods less radical than amputation. In keeping with this experience, the word *benign* was appended as a qualifying term in the name of the lesion. The similarity of certain characteristics of giant-cell tumor with commonly recognized reparative processes in bone cast doubt in some minds that any true neoplastic tendencies were involved. It is the present consensus that the term *giant-cell tumor of bone* should be restricted to designate a specific neoplastic lesion that usually is benign, has a tendency to recur and may, on occasion, present malignant characteristics at the outset or later in its course.

Giant-cell tumor of bone, representing something less than 20 per cent of all bone tumors, is not common. In the past 25 years, at the University Hospital, 48 examples have been seen. In this series there was no significant sex predominance, although others have reported that it is more common in females. This entity develops characteristically in young adults, usually after the age of 20, when epiphyses are fused. It arises usually in the ends of the long bones, probably in the epiphyses, and then extends to involve the adjacent metaphyses. In our experience, the sites most frequently involved have been the proximal tibia, the distal femur and the distal radius, in that order. A few authenticated examples have involved the spine, the pelvis and the mandible.

ROENTGENOLOGIC APPEARANCE

The classic roentgenologic appearance of a giant-cell tumor is a well-circumscribed cystic expanding lesion with thin trabeculae traversing the cystic space situated in one

* From the Department of Radiology, University of Michigan, Ann Arbor, Michigan



FIG. 1. Frontal and lateral projections of a giant-cell tumor of the distal fibula in a 15-year-old girl whose epiphyses are closed. The lesion is cystic, expanding and well demarcated, and contains thin trabeculae. It was excised, and a bone graft was inserted. Uncomplicated permanent healing resulted.

end of a long bone (Figs. 1 & 2). Characteristically, the adjacent joint is uninvolved, and no periosteal reaction is evident. Ordinarily, fracture occurs only in advanced lesions involving heavy weight-bearing lower extremity bones. Well-authenticated examples of giant-cell tumor have shown considerable variation from the usual roentgenologic pattern. Sometimes there is little or no bone formation within the lesion, the roentgenographic appearance being one of pure lysis. Histologically, such lytic lesions may show evidence of aggressive behavior, but this is not always the case. The lytic lesion shown in Figure 3 is somewhat less well defined than usual. Although giant-cell tumor is certainly to be considered in the differential roentgenologic diagnosis in this case, it is to be remembered that metastases from a distant primary are capable of producing similar appearances. When giant-cell tumor originates in the spine or the pelvis, the roentgenologic findings may be suggestive but scarcely characteristic. An example occurring in the spine and producing collapse of one vertebral body is shown in Figures 4 and 5.

It is important for a radiologist to have information regarding the history of an individual lesion before attempting to interpret film findings A previous biopsy, some form of surgical treatment or therapeutic irradiation, are all capable of modifying roentgenologic appearances, and unless known in advance the results of such procedures may be evaluated erroneously. It is important to



FIG. 2. Giant-cell tumor of distal radius in a 20-year-old man This roentgenogram shows the lesion after 2 recurrences. Although the characteristic lesion is large, the joint itself is uninvolved. Eventual healing occurred after excision, grafting and fusion of the radiocarpal joint.

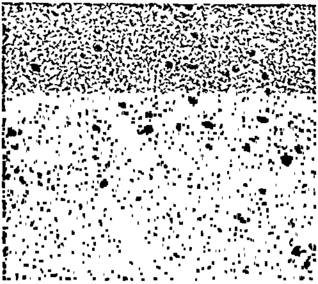


FIG. 6. Photomicrograph (× 95) of a giant-cell tumor that involved the proximal tibia in a 31-year-old man. The tumor is made up of a vascular stroma of uniform spindle-shaped cells with prominent nuclei. Scattered through this stroma are large multinucleated giant cells, whose nuclei are located centrally This lesion was curetted, bone chips were inserted, and healing progressed uneventfully. For 15 years there has been no further difficulty.

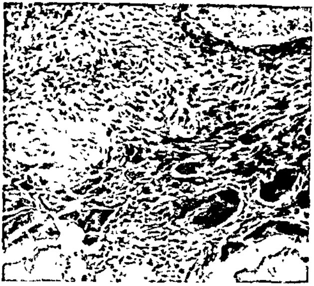


FIG. 8. Photomicrograph (× 285) of a peripheral portion of the lesion shown in Figure 1. This lesion was a giant-cell tumor, but in its periphery there were areas with considerable variation in appearance. In this one field a large cystic space, fibrous stroma, giant cells, bone resorption, some matrix and the cellular stroma of the giant-cell tumor are all visible. Some of the tissue elements present suggest a reparative process.

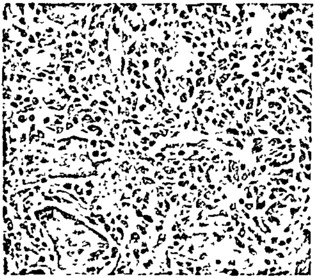


FIG. 7. Photomicrograph (× 335) of the giant-cell tumor shown in Figure 2. The histologic structure is that of a moderately cellular stroma of polyhedral cells with large, regular vesicular nuclei and little cytoplasm The large giant cells contain multiple nuclei that are quite similar to those in the stromal cells There is no matrix function

ities of these lesions have been responsible for considerable confusion in the matter of histologic and roentgenographic identification of giant-cell tumor. It is important to recognize this source of confusion and to segregate the giant-cell tumors from these so-called *variants*.

Ordinarily, giant-cell tumors have little, if any, extracellular substance or matrix. When, on histologic examination of material taken from a lesion considered to be a giant-cell tumor, considerable matrix is discovered, a degree of aggressiveness is suggested that is worrisome.

It is not always a simple matter to evaluate the growth behavior of a giant-cell tumor either from the roentgenographic or the histologic findings. The growth pattern may be completely benign, and even incomplete surgical removal or a small amount of radiation may result in healing with no tendency to recur. Such growth behavior is suggested and may be anticipated if the lesion presents



FIG. 9. Roentgenograms of an aggressive giant-cell tumor that had been present for 2 years in a 35-year-old woman. The films show extensive destruction and considerable involvement of the adjacent tissues. Histologically, the stroma of the neoplasm was very cellular. The limb was amputated, and the patient was alive and well 11 years later. (Hodges & MacIntyre: Am. Acad. Orthop. Surgeons Instructional Course Lectures VI, Ann Arbor, Edwards)

classic roentgenographic and histologic findings.

In a giant-cell tumor, poorly circumscribed bone lysis, excessive bone destruction or extension into adjacent tissues suggests aggressiveness that may be demonstrated by a tendency to recur or possibly to become malignant (Fig. 9). Histologically, aggressiveness is evidenced by increased cellularity of the stroma, variation in size and staining quality of the stromal cells, excessive mitotic activity and the presence of extracellular material (Fig. 10).

The evaluation of aggressiveness on the basis of histologic findings is relatively simple in the case of outright malignancy. The proper evaluation of microscopic findings in less clear-cut situations requires the services

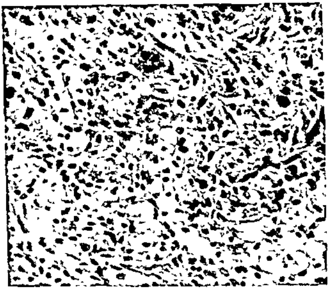


FIG. 10. Photomicrograph (× 335) of an aggressive giant-cell tumor in the distal radius of a 66-year-old man. The tissue shown came from the 3rd recurrence of this neoplasm, which was treated by amputation At this time the patient had pulmonary metastases. The neoplasm is composed of a cellular stroma with variation in size and in shape of the cells and of the staining qualities of the nuclei. The giant cells are small, but the nuclei are regular. Scattered foam cells are present. It would be very difficult, if not impossible, to say definitely that this lesion was malignant from a histologic study alone.

of an experienced pathologist, and at times it may be extremely difficult. The most effective method of evaluating aggressiveness in a given giant-cell tumor is to combine the opinions and the clinical experiences of the orthopaedic surgeon, the radiologist and the pathologist, rather than to rely upon any one viewpoint. Aggressive giant-cell tumors tend to recur, and recurrence is dependent to a considerable extent upon the type and the adequacy of treatment, as well as the biologic behavior of the neoplasm. Location of the lesion seems to be related to the tendency to recurrence. Those that develop in the distal portion of the radius are known to have a higher recurrence rate.

DIAGNOSIS

The accurate diagnosis of a giant-cell tumor involves the evaluation of clinical findings, roentgenologic appearance and histologic examination. It is our feeling that adequate biopsy is essential to establish the diagnosis, to help select the best method of treatment and to give some idea of the prognosis. For the best therapeutic results it is necessary that the variations in growth pattern and in the roentgenologic evidence of response to treatment be recognized and evaluated by a competent team of observers. Significant differences in ultimate outcome often depend upon proper attention to such details. Unless biopsy is employed as a routine diagnostic procedure, a variety of bone lesions may, on occasion, be erroneously considered to be giant-cell tumors and as a consequence subjected to ill-conceived treatment A list of situations that must be differentiated both roentgenographically and histologically includes traumatic bone cysts, fibrous dysplasia, eosinophilic granuloma, osteoid osteoma, the brown nodes of hyperparathyroidism, so-called giant-cell tumor tendon sheaths, subperiosteal hematoma, epulis of the mandible, and primary or metastatic malignant tumors. Of these, the one confused most commonly with giant-cell tumor of bone is the bone cyst Characteristically, this lesion occurs in children and in adolescents under the age of 20. It involves the metaphysis and is found to heal readily, even spontaneously, following fracture or minor surgical interference.

MANAGEMENT

Since the beginning of the century, with the acceptance of the idea that a giant-cell tumor of bone is not always a wildly growing process, treatment methods less drastic than amputation have been recommended and through usage have proved to be efficient. Curettage, cauterization and a combination of the two with the use of bone grafts have been recommended and subjected to clinical trial. In the light of experience there is little to indicate that cauterization, either chemical or electrical, offers any improvement over curettage. Destruction of the major portion of the neoplastic process by curettage, followed by insertion of bone chips or bone grafts that lend the supporting medium for reossification, has produced cure in a large number of patients. In many instances in which the giant-cell tumor involves a weight-bearing bone and a surgical approach would jeopardize seriously the function of the part, radiation methods of treatment have been found to be more feasible When lesions involve bones that are not primarily weight-bearing, total excision usually is the best method of treatment. Except in the case of those rare giant-cell tumors that exhibit frankly malignant characteristics, complete excision of the involved bone and its contiguous soft parts ensures a cure. Excision and bone grafting have proved to be efficacious on repeated occasions (Fig. 1).

Amputation still has a place in the management of giant-cell tumors of exceptional character when at the outset there is unquestionable evidence of aggressive growth involving a break through the cortex with involvement of soft tissues and adjacent bone structures (Fig. 8). In the series we have observed, amputation eventually was neces-

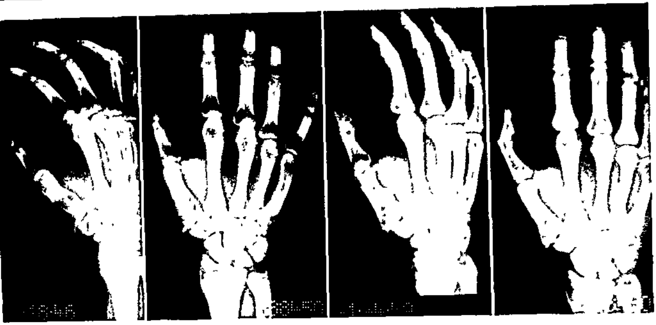


FIG. 11. Giant-cell tumor of the metacarpal in a 31-year-old man. The lesion was treated originally by curettage, but a recurrence developed 1 year later, and in June, 1946, this was the radiographic appearance. The lesion was treated with 1,200 r (air) to 1 field. Healing progressed satisfactorily, as is shown in the films made 2½ years later. Follow-up information indicates that the patient is well 5½ years after the irradiation.

sary in approximately one fifth of the patients. One half of the amputations were performed because of clinical signs of aggressive growth, nearly always confirmed by a corresponding histologic evidence. In 2 patients, osteomyelitis resulting from earlier and unwise surgical procedures led to amputation. In 1, amputation was necessary because an intercurrent fracture failed to heal. At times the traumatic, excessive irradiation employed in the early 1930's, before present-day concepts were in vogue, led to the ultimate necessity for amputation.

When first it became apparent that most giant-cell tumors could be controlled successfully by local methods of treatment, Pfahler and others demonstrated that irradiation was beneficial and could control the process. Prior to the late 1920's, inaccurate methods of measuring delivered radiation contributed heavily to the difficulties and the uncertainties encountered in some patients when this type of treatment was employed. In general, the earlier radiation treatment technics called for greatly excessive dosage, which often resulted in the loss of function of the part. We know now that moderate doses of external irradiation have proved to be efficacious in the treatment of many authentic giant-cell tumors (Fig. 11). When this type of radiation therapy is used, there follows frequently a period of lysis or so-called *osteolytic thrust,* which is followed in turn by subsequent reossification and healing. This is an expected reaction to the irradiation and should not be considered as evidence of aggressive behavior. In years past, some patients were cured apparently by the use of radium—by direct application, plaques or insertion of needles, or radon seeds. There is no reason to prefer this approach over external x-radiation.

Attempts to combine surgery and radiation have not been gratifying, despite the fact that there are recorded instances in which this procedure has succeeded. Far more often such combined measures have resulted in complications of one type or another. One of the chief sources of difficulty in the *combined approach* is the failure to recognize the postirradiation lysis and to proceed with some type of surgical treat-

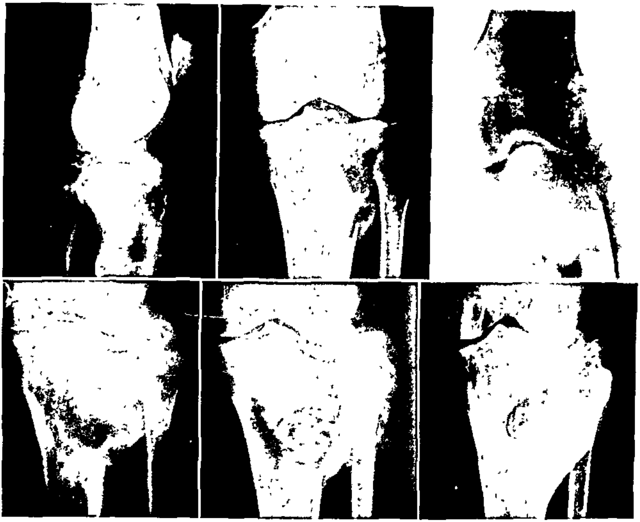


FIG. 12. Series of roentgenograms showing a largely lytic giant-cell tumor as it appeared first in November, 1945 (*top, left and center*). It was irradiated, 800 r (air) to each of 2 fields, and demonstrated the expected postirradiation lysis 3 months after treatment (*top, right*). Beginning recalcification is apparent 4 months after irradiation (Feb., 1946). *Unfortunately, bone chips were inserted in March, 1946 Note the progressing recalcification* of the cortex shown in April, 1946. Osteomyelitis developed, and was treated persistently over a 4-year period. (Hodges & MacIntyre: Am. Acad Orthop Surgeons Instructional Course Lectures VI, Ann Arbor, Edwards)

ment prematurely (Fig. 12). Such interference complicates both types of treatment, and there is a less satisfactory end-result than if either method were used alone. Postirradiation lysis becomes evident 4 to 6 weeks after irradiation and persists for a variable period of time up to 3 to 4 months. If the radiation method is employed, adequate time should be allowed for its effectiveness to be evaluated

Regardless of the type of treatment, the *possibility of recurrence must be anticipated*, and, if it occurs, further treatment, usually of a different type, is indicated In our material, radiation failures followed by surgical successes and surgical failures with ultimate radiation success are divided approximately equally. This does not include the small group of patients in whom there was evidence of aggressive growth unrestrained by either method.

There is no reliable method of predicting whether initially benign lesions ultimately will exhibit aggressive characteristics. If aggressive or frankly malignant characteristics are recognizable when the patient first is seen, it is not difficult to reach a decision in favor of radical treatment. In our series, there was

pathologic evidence of aggressive growth in 11 patients with corresponding clinical and roentgenologic evidence in 4 of the group. One patient showed unmistakable roentgenologic and clinical evidence of involvement of contiguous bone and soft tissue necessitating amputation, even though from the histologic viewpoint there were no clear-cut signs of aggressiveness. Another showed both clinical and pathologic evidence of aggressiveness when first seen, and in this instance amputation was performed immediately. Still another who was treated by both surgical and radiation methods failed to respond to either, and demonstrated both pathologic and clinical evidence of aggressiveness. Amputation in this instance was followed by no signs of recurrence or metastases.

In 2 patients the lesion proved to be progressive and resulted ultimately in death. In 1 of these there developed during the course of the disease a sufficient degree of cellularity within the stroma to represent true malignancy, and in this case distant metastases appeared (Fig. 9). In the other, who developed pulmonary metastasis and died ultimately, biopsy material from the original lesion has been reviewed by many authorities, none of whom has chosen to alter the original diagnosis of giant-cell tumor with only suggestive histologic evidence of aggressive behavior. There is little doubt that the aggressiveness of this particular lesion was underestimated and that the methods of treatment employed never were able to cope fully with the situation.

Although unmistakable examples of clinical or pathologic aggressiveness must be recognized and considered and evaluated carefully in planning and executing treatment, it would seem to be entirely unnecessary to consider that amputation is the treatment of choice for all patients in whom the diagnosis of giant-cell tumor is established. In the series reviewed, there have been numerous examples of giant-cell tumors so situated that surgical extirpation was not feasible and radiation methods have been successful in controlling the lesion over many years. Similarly, there have been examples of successful treatment by curettage and the insertion of bone chips. Either method may be expected to succeed unless clinical, roentgenologic or histologic appraisal discloses aggressive characteristics of the lesion. In view of numerous surgical and radiation successes, there is no apparent reason to abandon such procedures in favor of those that were considered wise at the turn of the century.

Our concept of a giant-cell tumor of bone has changed over the years. As more accurate information about the pattern of growth, the roentgenographic and the histologic characteristics, and the response to treatment becomes available, our concept of this lesion and our ability to diagnose and treat it adequately will increase.

Aspectos Radiologic de Tumores Ossee a Cellulas Gigante

Summario in Interlingua

Nostre concepto de un tumor ossee a cellulas gigante debe resultar del correlation de omne su attributos, su conducta biologic, su aspecto radiographic, su structura grossier e histologic, e su responsas a varie formas de therapia. Nostre concepto ha progredite, passante per varie phases, e nunc nos opina que iste lesion es neoplastic e que su formation characteristic occurre proxime al claudite linea epiphyseal de un osso longe in un juvene adulto. Il ha un certe variabilitate del aspecto radiographic e etiam del structura histologic. Le characteristic aspecto radiographic presenta un ben-demarcate lesion cystic que tende a expander se e contine tenue trabeculas, sed si le formation ossee es minimal o absente, il es possibile que le lesion presenta un aspecto predominantemente lytic e nonobstante es authenticamente un tumor a cellulas gigante.

Le methodos de tractar tumores ossee a cellulas gigante ha cambiate in le curso del evolution de nostre concepto del lesion.

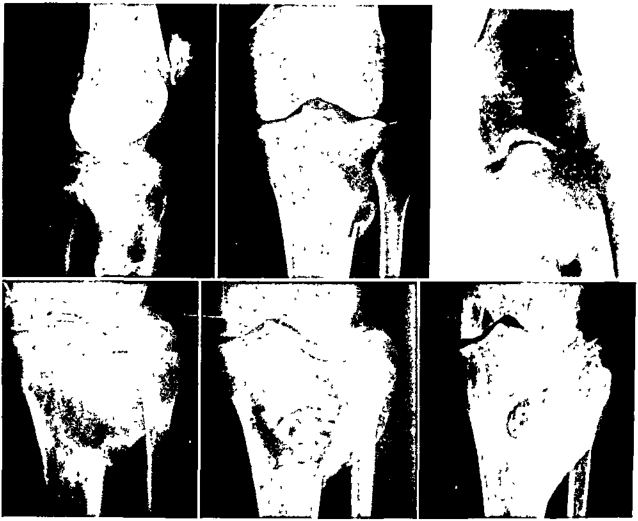


FIG. 12. Series of roentgenograms showing a largely lytic giant-cell tumor as it appeared first in November, 1945 (*top, left and center*). It was irradiated, 800 r (air) to each of 2 fields, and demonstrated the expected postirradiation lysis 3 months after treatment (*top, right*). Beginning recalcification is apparent 4 months after irradiation (Feb , 1946). Unfortunately, bone chips were inserted in March, 1946 Note the progressing recalcification of the cortex shown in April, 1946. Osteomyelitis developed, and was treated persistently over a 4-year period (Hodges & MacIntyre: Am Acad Orthop. Surgeons Instructional Course Lectures VI, Ann Arbor, Edwards)

ment prematurely (Fig 12). Such interference complicates both types of treatment, and there is a less satisfactory end-result than if either method were used alone. Postirradiation lysis becomes evident 4 to 6 weeks after irradiation and persists for a variable period of time up to 3 to 4 months. If the radiation method is employed, adequate time should be allowed for its effectiveness to be evaluated.

Regardless of the type of treatment, the possibility of recurrence must be anticipated, and, if it occurs, further treatment, usually of a different type, is indicated In our material, radiation failures followed by surgical successes and surgical failures with ultimate radiation success are divided approximately equally. This does not include the small group of patients in whom there was evidence of aggressive growth unrestrained by either method.

There is no reliable method of predicting whether initially benign lesions ultimately will exhibit aggressive characteristics. If aggressive or frankly malignant characteristics are recognizable when the patient first is seen, it is not difficult to reach a decision in favor of radical treatment. In our series, there was

pathologic evidence of aggressive growth in 11 patients with corresponding clinical and roentgenologic evidence in 4 of the group. One patient showed unmistakable roentgenologic and clinical evidence of involvement of *contiguous bone and soft tissue* necessitating amputation, even though from the histologic viewpoint there were no clear-cut signs of aggressiveness. Another showed both clinical and pathologic evidence of aggressiveness when first seen, and in this instance amputation was performed immediately. Still another who was treated by both surgical and radiation methods failed to respond to either, and demonstrated both pathologic and clinical evidence of aggressiveness. Amputation in this instance was followed by no signs of recurrence or metastases.

In 2 patients the lesion proved to be progressive and resulted ultimately in death. In 1 of these there developed during the course of the disease a sufficient degree of cellularity within the stroma to represent true malignancy, and in this case distant metastases appeared (Fig. 9). In the other, who developed pulmonary metastasis and died ultimately, biopsy material from the original lesion has been reviewed by many authorities, none of whom has chosen to alter the original diagnosis of giant-cell tumor with only suggestive histologic evidence of aggressive behavior. There is little doubt that the aggressiveness of this particular lesion was underestimated and that the methods of treatment employed never were able to cope fully with the situation.

Although unmistakable examples of clinical or pathologic aggressiveness must be recognized and considered and evaluated carefully in planning and executing treatment, it would seem to be entirely unnecessary to consider that amputation is the treatment of choice for all patients in whom the diagnosis of giant-cell tumor is established. In the series reviewed, there have been numerous examples of giant-cell tumors so situated that surgical extirpation was not feasible and radiation methods have been successful in controlling the lesion over many years. Similarly, there have been examples of successful treatment by curettage and the insertion of bone chips. Either method may be expected to succeed unless clinical, roentgenologic or histologic appraisal discloses aggressive characteristics of the lesion. In view of numerous surgical and radiation successes, there is no apparent reason to abandon such procedures in favor of those that were considered wise at the turn of the century.

Our concept of a giant-cell tumor of bone has changed over the years. As more accurate information about the pattern of growth, the roentgenographic and the histologic characteristics, and the response to treatment becomes available, our concept of this lesion and our ability to diagnose and treat it adequately will increase.

Aspectos Radiologic de Tumores Ossee a Cellulas Gigante

Summario in Interlingua

Nostre concepto de un tumor ossee a cellulas gigante debe resultar del correlation de *omne su attributos, su conducta biologic, su aspecto radiographic, su structura grossier* e histologic, e su responsas a varie formas de therapia. Nostre concepto ha progredite, passante per varie phases, e nunc nos opina que iste lesion es neoplastic e que su formation characteristic occurre proxime al claudite linea epiphyseal de un osso longe in un juvene adulto. Il ha un certe variabilitate del aspecto radiographic e etiam del structura histologic. Le characteristic aspecto radiographic presenta un ben-demarcate lesion cystic que tende a expander se e contine tenue trabeculas, sed si le formation ossee es minimal o absente, il es possibile que le lesion presenta un aspecto predominantemente lytic e nonobstante es authenticamente un tumor a cellulas gigante.

Le methodos de tractar tumores ossee a cellulas gigante ha cambiate in le curso del evolution de nostre concepto del lesion.

Ben que le majoritate del tumores ossee a cellulas gigante es benigne, il remane ver que illos tende a recurrer, e a vices illos es o deveni maligne. Le recognition del tendentias aggressive que resulta in recurrentia o malignitate es difficile e require le effortios cooperative de un equipa de competente investigatores. Varie formas de chirurgia e de therapia a radiation se ha provate capace a effectuar curas sin complicationes. In nostre experientia le combination de methodos chirurgic con methodos radiologic non se monstrava avantagiose. Quando on usa un therapia radiational on debe prestar attention al transiente lyse post-irradiational que se disveloppa ante le comenciamento del recalcification. Le area involvite in iste processo debe esser adequatemente protegite.

9

Aneurysmal Bone Cysts: Additional Considerations

BYRON E. BESSE, JR., M.D.,* DAVID C. DAHLIN, M.D.,†
DAVID G. PUGH, M.D.,‡ AND RALPH K. GHORMLEY, M.D.§

The term *aneurysmal bone cyst* first was used by Jaffe and Lichtenstein[12] in 1942 to designate a new and distinct entity. However, the lesion had been recognized prior to this time; it had been described as an ossifying hematoma as early as 1893.[17] In subsequent years it had been called by a multitude of conflicting and confusing names, including *atypical giant-cell tumor, pulsating benign giant-cell tumor, subperiosteal giant-cell tumor, central (medullary) giant-cell tumor, aneurysmal giant-cell tumor, hemorrhagic osteomyelitis, osteitis fibrosa cystica, benign bone aneurysm* and *angioma.*[1,2,4-7,9,10,15] Even as late as April, 1954, a review was made of 9 cases of so-called subperiosteal giant-cell tumors, with the suggestion that they also might be called *ossifying subperiosteal hematomas* or *aneurysmal bone cysts.*[16]

Here we wish to emphasize again the assertions of Jaffe,[11] of Lichtenstein,[13,14] and also of ourselves and associates,[3,8] that an aneurysmal bone cyst is a distinct clinical, roentgenologic and pathologic entity that is entirely different from a giant-cell tumor. Since our last presentation,[8] we have studied an additional 6 cases, including 2 from our old files, and are presenting here the statistics and the observations garnered from our total series of 32 cases.

* Fellow in Radiology, Mayo Foundation.

† Section of Surgical Pathology

‡ Section of Roentgenology.

§ Section of Orthopedic Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minn. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

CLINICAL ASPECTS

Incidence. Aneurysmal bone cysts constitute approximately 1.5 per cent of all primary tumors of bone removed surgically at the Mayo Clinic. In our experience, they are approximately one third as common as giant-cell tumors of bone.

Sex and Age. The age incidence in this series varied from 5 to 37 years, with an average age of 16 years; 24 patients were 19 years of age or less. This is in agreement with reports in the literature, in which the youngest patient described was 5 years of age and the oldest was 40.[11,13,14,16] It is of interest that this condition has not been reported in patients less than 5 years of age.

The female sex predominated by a ratio of 20 to 12 in our series. Lichtenstein[13] reported about an equal distribution between the sexes in 17 cases. Jaffe, in citing "about 20 cases," stated that females predominated; Thompson, in reporting 9 cases, noted an incidence of 8 males and 1 female. In a total of more than 70 cases thus far reported, the sex incidence is nearly equal.

Localization. The lesion may be found in practically any bone of the skeleton, with

a predominance in the long bones and the vertebrae. In our series, 7 lesions were in tibiae, 5 in fibulae, 4 in femora, 1 in the humerus, 2 in ulnae and 6 in vertebrae. This total includes 25 of the 32 lesions, or approximately 80 per cent. The remaining 7 lesions were as follows: 2 in the ilia, 1 in the ischium, 1 in the patella, 1 in a rib, 1 in the occipital bone and 1 in the sternum. These cysts have been reported also in the pubis, the clavicle, the metacarpal, the phalanx, the os calcis, the talus and the tarsal navicular; 1 lesion involved multiple bones of the foot.[11,13,16] The only parts of the skeleton in which aneurysmal bone cysts have not been reported are the facial bones and the mandible, and the remaining bones of the calvarium other than the occipital bone.

Symptoms and Signs. The most frequent complaints are localized pain and swelling. These were present in 28 of our cases; usually they occurred together. Ordinarily, the pain was a dull, aching one that increased with exercise. The swelling was insidious in onset and of a slowly progressive nature. Tenderness occurred in 19 cases and was localized to the area of swelling. Occasionally, redness was associated with the tenderness. Limitation of motion occurred in 15 cases, being associated with those lesions whose mass interfered with normal motion of the joint. Signs of involvement of nerve



Fig. 1. (*Left*) Typical lesion involving the proximal part of the shaft of the right humerus. Eccentric bulging of the lateral cortex is present, with a thin outer shell of delimiting new bone and coarse to fine trabeculae. The lesion has involved the entire width of the shaft, with slight erosion of the cortex medially. (*Right*) Poorly outlined aneurysmal bone cyst involving the shaft of the distal portion of the left femur. Ossification of the epiphysial plate is present, and the lesion extends into the lateral condyle.

roots or the spinal cord occurred in 2 of the cases in which vertebral involvement was present. The duration of symptoms varied from 3 weeks to 4 years, the average being 7 months.

ROENTGENOLOGIC FINDINGS

The characteristic findings are seen most frequently in the large long bones and the vertebrae, and consist of an eccentric ballooned-out appearance of the cortex with extension of the mass of the lesion into the surrounding soft tissues. The mass is surrounded by a thin layer of cortex or periosteal new bone. Trabeculae are present; they are coarse at the periphery of the lesion and become more delicate toward the center. The lesion has an interior radiolucency. These findings are most suggestive of an aneurysmal bone cyst (Fig. 1, *left*). At the junction of the normal and the involved portions of the bone, multiple calcified layers of periosteal new bone may be seen, giving the so-called onion-peel appearance indicative of the slowly progressive nature of the lesion. In our series, the cyst invariably arose in the diaphysis. If endochondral bone growth was occurring, the lesion always was on the diaphysial side of the epiphysial plate. Only in older patients, after the epiphysial cartilage had ossified, did the lesion extend into the epiphysis (Fig. 1, *right*).

As already indicated, the vertebrae were involved in 6 of our cases. In all cases, excepting the 1 in which the lesion was in the sacrum, the roentgenologic appearance was typical of aneurysmal bone cyst. In these 5 typical cases, the lesion arose in the vertebral body in 3 cases, in the transverse process in the fourth and in the spinous process in the fifth It extended to produce pressure erosion of the contiguous bony structures in all 5 cases, and all 5 exhibited the typical ballooned-out appearance with extension of the tumor into the soft tissues. The mass was surrounded by a definite outer shell of thin bone, and the usual trabeculae were present—coarse at the periphery and more delicate toward the center. The interior of all the lesions was radiolucent. The sixth lesion arose in the body of the third sacral vertebra on the right and eroded the body of the fourth sacral vertebra on the right by continuity; the main portion of the tumor presented anteriorly. A thin, interrupted cortex could be seen, with a suggestion of trabeculae. However, the lesion could not be diagnosed definitely as an aneurysmal bone cyst in the roentgenogram. In no case has the lesion appeared to be primary in more than 1 vertebra. In a series of 101 proved giant-cell tumors studied at the clinic by Williams and his associates,[18] none has occurred in the vertebrae other than a few in the sacral segments.

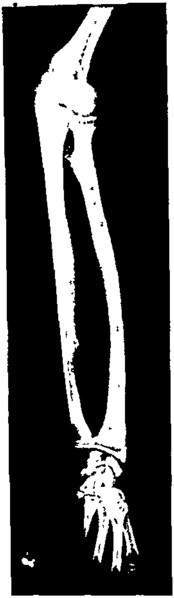


FIG. 2. Aneurysmal bone cyst involving the shaft of the left ulna, near the junction of the middle and the distal thirds. This is the only cyst of its kind seen in a long bone that did not occur at the end of the diaphysis.

When the lesions are in flat or slender



FIG. 3. Aneurysmal bone cyst of the right patella with slight expansion of the entire bone; note intact outer layer of cortex and coarse trabeculae.



FIG. 4. Aneurysmal bone cyst involving the right ischium. A thin outer rim of periosteal new bone can be seen inferiorly. A large soft tissue mass extends superiorly into the pelvis without evidence of trabeculation or delimiting outer shell of bone.

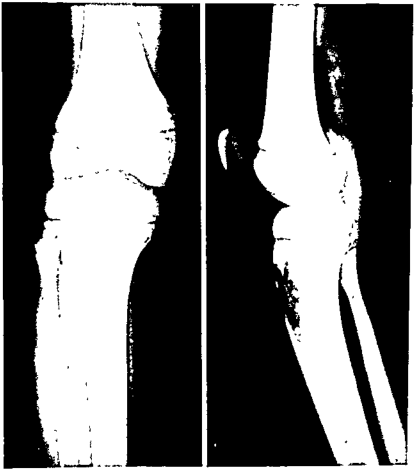


FIG. 5. Aneurysmal bone cyst with involvement of the shaft of the proximal part of the right tibia The cortex has not bulged eccentrically, and only a zone of radiolucency can be seen, with a few scattered trabeculae.

tubular bones and when the lesions are early or far advanced, the findings may be less characteristic but still suggestive of aneurysmal bone cyst. Those occurring in flat or slender tubular bones show an occasional eccentric blowout, but more frequently a symmetric dilatation of the involved shaft occurs. The cortex is greatly thinned on all sides, and the lesion extends into the soft tissues but is outlined by a thinned cortex or periosteal new bone. In our experience, trabeculation always was in evidence in the flat bones, and more often than not it extended across the entire bone, being more delicate toward the center. Multiple layers of periosteal elevation were seen invariably at the junction of normal and abnormal bone (Figs. 2-4).

In the early lesions, especially of large tubular bones, all one may see is an area of radiolucency, usually in the end of the diaphysis, without trabeculation or bulging of the cortex (Fig. 5). These lesions can be differentiated roentgenologically from unicameral cysts of bone in that the latter usually have sharper and more distinct margins. However, in our experience, all but 1 aneurysmal bone cyst involving a long bone occurred adjacent to the epiphysial plate in the diaphysis and did not migrate, as unicameral cysts of bone do characteristically with growth. The radiolucent appearance alone has been seen in 2 tibial lesions involving the upper portion of the shaft.

In far-advanced lesions the mass may attain tremendous size and bulge in all directions (Fig. 6). Nevertheless, remnants of cortex or periosteal new bone formation or both still persist, especially near the junction of normal and abnormal bone. Sparse trabeculae are seen.



FIG. 6. Aneurysmal bone cyst involving the proximal part of the shaft of the right ulna. Note the multiple layers of periosteal reaction and the well-delineated outer margin.

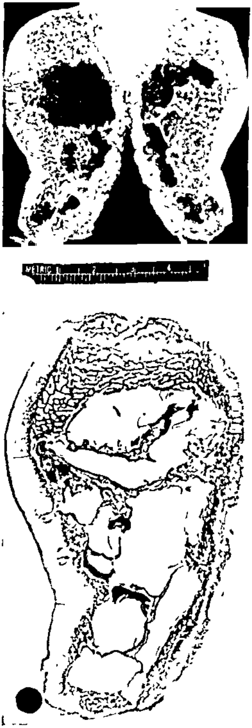
PATHOLOGIC FINDINGS

The pathologic features of aneurysmal bone cysts have been discussed previously in detail.[8,14] We shall merely summarize the salient features seen in the gross specimens available for review in all our cases. We were aided greatly in that 10 of these specimens were intact, total excision or amputation having been done. Orientation may be difficult if one is reconstructing curetted fragments. Blood-filled spaces of various size constituted the bulk of the lesion in each case. The centrally located spaces may be several centimeters in diameter. The smaller ones, often more numerous peripherally, varied in size from a few millimeters down to those visible only microscopically. Septa may be veil-like and translucent or up to a centimeter or more in thickness; they may be soft and friable or they may be fibrous; they often contain macroscopic and microscopic blood spaces (Fig. 7). Noncavernous soft or solid areas may constitute nearly half the bulk of unusual lesions. Beneath the delimiting bulged periosteum is a layer of bone, ordinarily of eggshell thickness. Bound-



FIG. 7. Unroofed aneurysmal bone cyst of cervical vertebra, showing cavernous spaces separated by septa.

FIG. 8. (*Top*) Typical lesion of patella; this is the lesion seen roentgenologically in Figure 3. The blood has run out of the cavernous spaces. (*Bottom*) Section of entire patella, showing the abrupt transition to normal bone; the darker portions of the septa contain numerous giant cells (hematoxylin and eosin; × 2).



aries between the lesion and adjacent bone are discrete, with normal bone abutting on the lesion.

Unclotted blood filled the cavernous spaces in all these lesions (Fig. 8, *top*). Bleeding often was a prominent feature at operation; the blood welled up into the lesion but did not spurt from it. A surgeon encountering these characteristic features should strongly suspect the correct diagnosis.

The microscopic finding of cavernous spaces or portions of them confirms the correct diagnosis (Fig. 8, *bottom*). The walls of these spaces do not contain the elastic lamina and muscle or lining endothelium seen in blood vessels The walls (septa) are composed of fibrous tissue, often containing long thin bands of osteoid (Fig. 9). Occasional or numerous multinucleated benign giant cells may be present, especially in the thicker septa or in solid parts of the lesions. These giant cells are the type seen in a wide variety of bony lesions, and they should not be construed as an essential part of the pathologic changes in aneurysmal bone cysts (Fig. 10) The natural tendency to take microscopic sections from the solid zones has helped in the confusion of aneurysmal bone cysts with giant-cell tumors. In some lesions, rather prominent osteoid plaques are seen in the solid zones, in others, relatively



FIG. 9. Curetted fragments. Collapse of many spaces has occurred; the thin strands of osteoid in the septa are prominent (hematoxylin and eosin; × 35).

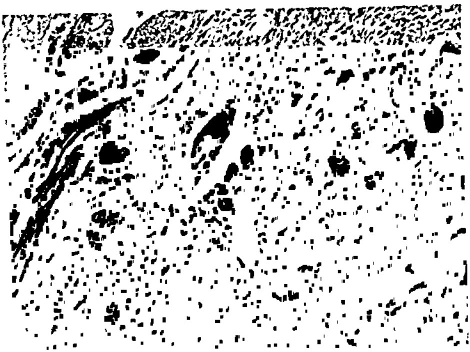


FIG. 10. Fibrous area containing benign multinucleated cells; such a field is not specific for aneurysmal bone cysts (hematoxylin and eosin; × 120).

acellular fibrous tissue is found in the solid zones. Phagocytes, lymphocytes or extravasated blood may be found in the septa.

RESULTS OF TREATMENT

Follow-up data are available for periods ranging from 4 to 35 years with regard to 23 of these patients. Of the remainder, 6 have been treated within the past 3 years and 3 did not respond to follow-up letters. Of the 23 patients followed for 4 years or more, 6 had complete excision of the affected portion of bone (4 lesions of the fibula, 1 of the spinous process of a cervical vertebra and 1 of a rib). All these patients were cured. Thirteen patients had thorough curettage, with only 2 instances of recurrence in this group. One of these 2 patients had 2 recurrences in 3 years, and further follow-up is necessary to ascertain the ultimate outcome; the other patient had roentgenologic evidence of a small recurrent lesion 6 months after the original curettage and postoperative radiation, but this disappeared after additional radiation. Previous partial removal or "exploration" had proved to be ineffectual

in 4 of this total of 19 patients having either total excision or thorough curettage. Twelve of these 19 patients, including the 2 with recurrent lesions, had variable but relatively little adjunctive radiation therapy. There was no instance in which the lesion regressed after biopsy alone.

Amputation was performed in the remaining 4 of the 23 patients followed 4 years or more. In 1, it was necessary because of the size of the lesion, which had destroyed the upper half of the tibia. In 2, amputation followed an erroneous diagnosis of a malignant lesion. In the fourth patient, amputation was necessary 3½ years after exploration followed by radiation treatment elsewhere. This last lesion, which was in the upper portion of the tibia, had progressed despite 3 courses of roentgen therapy and 2 surgical attempts at removal.

DIFFERENTIAL DIAGNOSIS

Despite its distinctive features, the relative rarity of this bony lesion has made its general recognition slow. Once the physician is familiar with the disease, the diagnosis is made readily.

Aneurysmal bone cysts are confused most commonly with giant-cell tumors of bone, but in the average case the pathologic distinction should be easy. The cavernous spaces necessary to the diagnosis of aneurysmal bone cysts are lacking in giant-cell tumors In the diagnostic zones of a giant-cell tumor, the giant cells lie among stromal cells that have practically no intercellular substance. Ordinarily, the solid zones containing these multinucleated cells in aneurysmal bone cysts are more fibrous or contain abundant osteoid. The differentiation of these 2 lesions cannot always be made on a single microscopic section from each tumor, but reference to the gross specimen and procurement of appropriate new sections solve the problem readily However, we have observed 1 typical giant-cell tumor for which curettage and bone grafting were done; on recurrence, this lesion contained zones similar to those seen in an aneurysmal bone cyst. Whereas, in our experience, 90 per cent of giant-cell tumors have been in patients past the second decade of life, 75 per cent of the patients who had aneurysmal bone cysts were less than 20 years of age. Furthermore, some of the latter lesions were found in bones such as the vertebrae and the skull, where giant-cell tumors practically never occur. Although about 10 per cent of giant-cell tumors become manifestly malignant, such a change has not yet been observed in aneurysmal bone cysts in our experience. Furthermore, the rate of recurrence is much greater in giant-cell tumors than in aneurysmal bone cysts. Giant-cell tumors occur most frequently in the epiphysis and do not show the roentgenologic findings of the eccentric blowout, the outer thin shell of bone or the trabeculation characteristic of aneurysmal bone cysts

Simple unicameral cysts of bone should not be confused with aneurysmal bone cysts. We have observed occasional simple cysts with partial septa that macroscopically and microscopically were similar to those of aneurysmal bone cysts, but these contained serous fluid instead of blood. Ordinarily, the lining of a unicameral cyst of bone is a thin layer of fibrous tissue, and, although benign giant cells may be numerous, there are no other histologic similarities to aneurysmal bone cysts. The lesions are dissimilar roentgenologically in that unicameral cysts of bone do not show the characteristic findings of an aneurysmal bone cyst. Even in early aneurysmal bone cysts confined to the medullary cavity, there is an indefinite, less distinct margin than is seen ordinarily with a unicameral cyst.

COMMENT

The pathogenesis of aneurysmal bone cysts still is unknown. It appears reasonable to consider that this process is not neoplastic but apparently is due to some obscure local circulatory disturbance.[13] Only 15 of our 32 patients gave histories of trauma, much of it minor. As does Lichtenstein,[13] we consider it unlikely that injury produces the lesion

The fact that these lesions do not follow fractures or surgical procedures supports this view. A history of trauma is a common finding in many tumors of bone, in which apparently it calls attention to the lesion rather than acts as a causative factor.

The histories in our cases support Lichtenstein's[13] observation that aneurysmal bone cysts enlarge progressively until adequate treatment is instituted. We have had the opportunity to follow the progress of several of these lesions on roentgenograms in cases in which operation at first was refused. In no instance had the lesion regressed spontaneously, and in 2 cases the lesions attained such tremendous size that amputation was necessary for surgical correction. One of these cysts involved the entire upper third of the tibia and produced erosion of the fibular head, loss of joint space and erosion of the distal femoral condyles.

Although one author stated[16] that some of the lesions in his series began in the soft tissues and later involved bone, we had no cases in which the cysts were not obviously primary in bone. Indeed, several appeared to have begun in the medullary cavity and involved the cortex secondarily.

Some authorities have advanced the theory that an aneurysmal bone cyst is an organizing zone of hemorrhage.[15] If this were true, it would not appear to be possible for the lesion to progress slowly in size and to contain regions of nonstagnant blood with honeycombed spaces. It has been suggested also that these cysts may be a modification of some other lesions of bone.[10] However, neither Lichtenstein,[14] in more than 30 cases, nor we have observed tangible evidence of a pre-existing lesion.

Although we have not encountered the development of sarcoma as a complication of aneurysmal bone cysts, a word of caution may be in order. Lichtenstein[13] studied 1 patient whose lesion involved the lower part of the femur and who was treated with roentgen rays, receiving a tumor dose estimated at 2,600 r. A fibrosarcoma developed at the same site 6 or 7 years later, and, despite amputation, the patient succumbed to widespread visceral and skeletal metastasis. It is possible, of course, that this sarcoma was produced by the treatment.

SUMMARY

The findings in a study of 32 aneurysmal bone cysts at the Mayo Clinic further confirm the identity of this lesion. The clinical, the roentgenologic and the pathologic features indicate that aneurysmal bone cysts are a distinct benign entity, and that in no sense is the lesion a variant of giant-cell tumors of bone. The roentgenologic findings are typical in two thirds of the cases, and the gross and the microscopic appearance of the lesions confirms the diagnosis readily. The treatment of choice is complete excision, if practicable, or thorough curettage. The incidence of antecedent trauma probably is no greater than it is in any other bony lesion.

REFERENCES

1. Adson, A. W.: Osteitis fibrosa cystica of the spine, Surg., Gynec. & Obst. 46:684, 1928.
2. Barrie, George: Hemorrhagic osteomyelitis, J. Bone & Joint Surg. n.s. 4:653, 1922.
3. Besse, B. E., Jr., Dahlin, D. C., Bruwer, André, Svien, H. J., and Ghormley, R. K.: Aneurysmal bone cyst, Proc. Staff Meet., Mayo Clin. 28:249, 1953.
4. Bloodgood, J. C.: Bone tumors, benign bone cysts due to central ostitis fibrosa of the unhealed latent type: with discussion of the operative treatment, especially the crushing of the cyst wall, J. Radiol. 4:345, 1923.
5. ———: Bone Tumors: Central (medullary) giant-cell tumor (sarcoma) of lower end of ulna, with evidence that complete destruction of the bony shell or perforation of the bony shell is not a sign of increased malignancy, Ann. Surg 69:345, 1919.
6. Coley, B. L., and Miller, L. E.: Atypical giant cell tumor, Am. J. Roentgenol. 47: 541, 1942.
7. Cone, S. M.: Ossifying hematoma, J. Bone & Joint Surg. n s. 10:474, 1928.
8. Dahlin, D. C., Besse, B E., Jr., Pugh, D. G., and Ghormley, R. K.: Aneurysmal bone cysts, Radiology (in press).

9. Ewing, James: Neoplastic Diseases: A Treatise on Tumors, ed. 4, pp. 323-324, Philadelphia, Saunders, 1940.
10. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, ed. 3, pp. 272-273, 309-316, 614-619, Philadelphia, Lippincott, 1949.
11. Jaffe, H. L.: Aneurysmal bone cyst, Bull. Hosp Joint Dis. 11:3, 1950.
12. Jaffe, H. L., and Lichtenstein, Louis: Solitary unicameral bone cyst: with emphasis on the roentgen picture, the pathologic appearance and the pathogenesis, Arch. Surg. 44:1004, 1942.
13. Lichtenstein, Louis: Aneurysmal bone cyst: further observations, Cancer 6:1228, 1953.
14. ———: Aneurysmal bone cysts: a pathological entity commonly mistaken for giant-cell tumor and occasionally for hemangioma and osteogenic sarcoma, Cancer 3: 279, 1950.
15. Shallow, T. A., and Wagner, F. B., Jr.: Pulsating benign giant cell tumors of bone: report of a case and review of the literature, Arch. Surg. 52:661, 1946.
16. Thompson, P. C.: Subperiosteal giant-cell tumor: ossifying subperiosteal hematoma —aneurysmal bone cyst, J. Bone & Joint Surg. 36-A:281, 1954.
17. Van Arsdale, W. W.: Ossifying haematoma, Ann. Surg 18:8, 1893.
18. Williams, R. R., Dahlin, D. C., and Ghormley, R. K: Giant-cell tumor of bone, Cancer 7:764, 1954.

Cystes Ossee Aneurysmatic

Summario in Interlingua

Esseva studiate un serie de 32 casos de cyste ossee aneurysmatic. Le etate del patientes in iste serie variava ab 5 a 37 annos. Le etate median esseva 16 annos. Le distribution sexual esseva 20 femininas e 12 masculos. Le lesion occurreva in omne ossos del corpore, excepte le ossos facial. Le aspectos roentgenologic esseva typic in circa duo tertios del casos e presentava un tenue concha *de nove osso periosteal delimitante* le peripheria del lesion, un configuration de bullas de sapon o de faveolos al radiolucente interior del lesion, e un eccentric protrusion del cortice que esseva generalmente disrumpite. Le typic aspectos pathologic consisteva in omne caso de ingorgate spatios vascular de varie dimensiones e characterisate per parietes sin le tractos de normal vasos sanguinee, septos de texito conjunctive que frequentemente includeva tenue bandas de osteoide (osso fibrose), e accumulationes de non-coagulate erythrocytos in le spatios vascular. Le therapia de election es excision complete, si practicabile, o curettage minutiose. Le prognose es excellente. In nostre serie il habeva solmente un caso de recurrentia del lesion, e isto esseva apparentemente curate per un secunde intervention operative. Le frequentia de antecedente traumas es probabilemente non plus alte que in le caso de altere lesiones ossee. Nostre studio corrobora le constatation de Jaffe e Lichtenstein que un cyste ossee aneurysmatic es un distincte entitate clinic, roentgenologic, e pathologic e non deberea esser confundite con ulle altere lesion ossee.

10

Chordoma*

THOMAS F. MEANEY, M.D.,† CHARLES M. GREENWALD, M.D.
AND GEORGE S. PHALEN, M.D.

Chordomas are rare neoplasms of low grade malignancy that originate from the neoplastic proliferation of notochordal remnants in the nucleus pulposis, as well as from aberrant vestiges within the skull and the vertebrae. Their course is marked by slow expansile growth with destruction of bone, extension into adjacent structures and recurrence after surgery. Although rare, chordomas must be considered in the differential diagnosis of many lytic lesions of the spine. Patients having chordomas of the sacrum or of any other portion of the spinal column often consult an orthopaedist because of pain in the back or in the neck. It is our purpose in this chapter to review the tumor briefly, including the etiology and the pathogenesis, and to present representative case histories of the course of chordoma in various locations.

The notochord is a column of cells extending from the buccopharyngeal membrane to the coccyx that forms the primitive embryonic axial skeleton of all vertebrates. As the vertebrae and the intervertebral disks develop, undifferentiated mesenchymal elements surround the notochord. The notochord becomes segmented, and the vertebral portions disappear. In the intervertebral disks, there is normal persistence of notochordal tissue in the form of the nucleus pulposis.[1,14] Notochordal rests, however, may persist anywhere along the craniospinal axis, and it is from these aberrant vestiges that chordomas develop.

These tumors have a somewhat typical gross appearance. They present an encapsulated, partially translucent grayish-to-blue mass with a liquid or gelatinous content, with areas of hemorrhage and calcification. In 1952, Dahlin and MacCarty[3] presented an excellent review of the histopathology of chordomas. The histopathologic features of chordomas are as follows (Fig. 1): a lobular arrangement of the cells, intracellular mucus in the form of cytoplasmic vacuoles, areas of physaliphorous cells, nuclear vacuoles, and cytoplasm that may form a syncytium with indiscernible individual cell bodies and nuclei throughout. Extracellular mucus was a constant finding. The presence of mitotic figures and anaplasia did not affect adversely the length of survival in the cases that they studied. Although chordomas may arise from notochordal vestiges anywhere along the craniospinal axis from the sella turcica to the coccyx, there is a marked predilection for the extremes of this axis. A comprehensive review of 252 chordomas by Faust, Gilmore and Mudgett[5] revealed the following distribution: 48 per cent sacrococcygeal, 37 per cent cranial, 13 per cent vertebral and 2 per cent eccentric. Eccentric locations include the mandible, the maxilla and the superior portion of the occipital bone.

* From the Departments of Roentgenology and of Orthopedic Surgery, The Cleveland Clinic Foundation, and The Frank E. Bunts Educational Institute, Cleveland, Ohio.

† Fellow in Department of Roentgenology, Cleveland Clinic.



FIG. 1. Histologic section (× 200) of a chordoma showing vacuolated cells arranged in interlacing cords and separated by a myxomatous stroma.



FIG. 2. Axial projection showing extensive erosion of the left petrous apex as compared with the intact right side. A large tumor mass fills the middle fossa.

A total of 19 chordomas have been seen at the Cleveland Clinic during the period 1929 to 1955, 4 of which have been reported previously.[4,8] The unusual feature in our series is the preponderance of cranial and vertebral lesions, particularly the latter. There were only 4 sacrococcygeal chordomas, an incidence of 21 per cent, as compared with 9 cranial and 6 vertebral tumors, forming respectively 47 per cent and 32 per cent of the total series All 6 vertebral tumors and only 1 sacral lesion have been found since 1951, while all 9 of the cranial tumors and the remaining 3 sacral tumors were found prior to this. This suggests the possibility that with improved diagnostic technics and the greater emphasis on definitive diagnosis in recent years, an increased number of chordomas will be found in the vertebral location Such lesions may previously have been misdiagnosed clinically as any one of a number of more common growths that chordoma may simulate.

Chordomas may be found in persons of any age. Although the finding has been reported in infants and even in a fetus, the greatest incidence occurs in the middle decades. A definite discrepancy is apparent in the average age of patients having the cranial lesions as compared with that of patients with chordomas in other locations. The average age at surgery of our 9 patients with cranial tumors was 36 years (range, 21-59 years). The average age of those having sacrococcygeal or vertebral lesions was 54 years (range, 30-68 years). This earlier onset of cranial chordoma by more than a decade has been noted previously by other authors.[5,9] It cannot be easily attributed to a more rapid growth of the cranial tumor, since the duration of symptoms is essentially the same, regardless of the location. The sex incidence was about evenly divided, there being 10 females and 9 males. This is at variance with the accepted higher incidence in males in the ratio of 2:1.

CRANIAL CHORDOMA

The chief characteristic of chordoma in all locations is slow growth with symptoms of long duration, often years rather than months. A correct preoperative diagnosis is uncommon because there are no pathognomonic clinical findings. Cranial chordomas arise most commonly from the region of the clivus at the spheno-occipital synchondrosis, but they can occur from the sella turcica to the anterior margin of the foramen magnum. Patients present definite neurologic signs and symptoms of intracranial lesion. The most common complaints in our series were headache and visual disturbance with diplopia from involvement of the 6th cranial nerve. The duration of symptoms ranged from 1 month to 5 years, averaging 18 months. Findings depend on the size of the tumor and the direction of its growth; most commonly its direction is posterior with involvement of the pons and the medulla.[8] Anterior extension to the sella and the parasellar structures is not uncommon, and advanced lesions may involve the sphenoid sinus, the nasopharynx, the maxilla and the orbit.

Early bone changes resulting from cranial chordoma may not be apparent roentgenologically on routine skull films; they may be demonstrated only on an axial projection for visualization of the base of the skull (Fig. 2). Osteolytic destruction of the basisphenoid is the rule, and the destruction may extend to the petrous apex, the floor of the middle fossa or the sella turcica. In Wood and Himadi's series,[15] calcified particles were demonstrated frequently in the region of the clivus, possibly as a result of inclusion of sequestrated bone fragments in the chordoma or degenerative changes in the neoplasm. A large calcified intracranial mass with only a central zone of tumor remaining has been reported by Freeman.[6] Roentgen changes caused by cranial chordomas usually cannot be differentiated from those caused by other neoplasms occurring in this region. The differential diagnosis includes glomus jugularus tumor, tumors of the pituitary, acoustic neuroma, craniopharyngioma and extension from carcinoma of the sphenoid sinus and the nasopharynx.

Case 1. This 40-year-old woman was admitted to the Cleveland Clinic Hospital on January 12, 1946, because of pain in the left ear, left-sided headache, neck pain and diplopia. The symptoms had begun approximately 18 months previously. Approximately 2 weeks before admission she developed severe pain in the left tonsillar region, followed by numbness of the left side of the face and the tongue. One week later a convulsion occurred.

The neurologic examination revealed diminished vision in the left eye, choked disks with hemorrhage on the left side, paralysis of the 6th nerve, diplopia, nystagmus on the right lateral gaze, numbness of the left side of the face and the left side of the tongue, absent left corneal reflex, diminished hearing in the left ear, diminished gag and palatine reflexes and suboccipital tenderness.

The only abnormal finding on routine laboratory study was 40 mg. of protein in the spinal fluid. Roentgen examination of the skull revealed a destructive lesion, osteolytic in character, involving the apex of the left petrous ridge on basilar view (Fig. 2).

On left suboccipital craniotomy a bluish cystic mass was found lateral and just posterior to the internal acoustic meatus in the region of the jugular foramen. When opened, the mass contained a large quantity of semigelatinous, clear tan-colored material. Pathologic diagnosis was chordoma.

Eleven months after operation the patient was relatively asymptomatic, but she had impairment of the left 5th, 8th and 9th cranial nerves, and nystagmus on lateral gaze.

Approximately 4½ years postoperatively the patient noted marked tinnitus, further diminution in the defective hearing on the left with a gradual onset of dysphagia. In addition, there had been blurring of vision and diplopia, as well as hoarseness.

A second craniotomy was performed on May 11, 1951. Anterior to the cerebellum a bulging mass, apparently encapsulated, was found. A large quantity of the tumor was removed. Pathologic diagnosis again was chordoma. The patient was discharged from the hospital and subsequently was lost to follow-up.

SACROCOCCYGEAL AND VERTEBRAL CHORDOMAS

The duration of symptoms for the sacrococcygeal and the vertebral lesions in our



Fig. 3. Lateral photograph of patient shows an extremely large soft tissue mass projecting downward from the caudal region.

series ranges from 3 months to 6 years, averaging 18 months. Pain usually is the main complaint. It often is sharp and radiating as a result of involvement of the nerve roots. When the tumor encroaches on the cord, symptoms are those of neoplasm of the spinal cord, with paralysis and sensory changes.

A special feature of the sacrococcygeal tumors is the very large tumor mass that may be present The initial symptoms may be rectal or urinary, due to the encroachment on these organs by anterior growth of the tumor into the pelvis Obstetric disproportion has been observed due to a large antesacral chordoma.[7] An unusually large posterior caudal mass was present in one of our cases (Fig. 3).

Although not diagnostic, the roentgen ap-



Fig. 4. Roentgenogram of pelvis. A large multiloculated osteolytic mid-lin defect, measuring some 7 cm., is present i the lower sacral region.

pearance of chordomas in the sacrococ eal area comes the closest to being cl acteristic. A large irregular osteolytic n line defect is seen with well-defined mar due to slow growth, but without marg sclerosis (Fig. 4). There usually is an sociated soft tissue mass. Rarely, the m may predominate with no evidence of b destruction on roentgenograms. The difl ential diagnosis includes teratoma, giant- tumor, chondrosarcoma, extension fr pelvic and rectal neoplasms, and metast malignancy.

Case 2. This 72-year-old white male first seen on August 1, 1951, with the complaint a large swollen area of induration over the buttock of 2 to 3 months' duration.

Physical examination was essentially wit normal limits except for one plus ankle ede and findings in the buttock region. Over left buttock was a large mass, rubbery in c sistency, indurated, and lying deep in the l buttock. It extended to the mid-line and bel into the posterior thigh. Roentgen examinati

of this patient revealed a large soft tissue mass posterior to the sacrum. No bony change was demonstrable. This mass was biopsied, and the pathologic diagnosis was chordoma. Removal was not attempted.

Approximately 18 months later the patient was seen again; the mass was much larger than it had been and occupied the entire area of the left buttock, including most of the posterior thigh (Fig. 3). The patient was asymptomatic except for a dull ache when he sat. This mass appeared to contain a large amount of fluid, and aspiration yielded a mucoid, odorless brown fluid. The patient underwent surgery on April 15, 1953. A trocar was introduced into the tumor, and 1,500 cc. of a brownish mucoid material was aspirated. The tumor was found to extend down into the gluteal muscles, and large portions of the tumor were removed. Because of the large size and uncontrollable bleeding at the time of operation, no attempt was made to remove the entire mass.

The patient's symptoms improved, and 4 months later the mass had not recurred and the patient was doing well, free of pain. However, by February, 1954, the mass had recurred and was as large as, or larger than, it had been prior to surgery in 1953.

No further follow-up has been obtained.

Case 3. A 44-year-old male first was seen at the Cleveland Clinic because of pain in the low back region and in the mid-line that had begun 9 months previously. Since its onset, the pain had been persistent, and deep and aching in character. Subsequently, the patient had experienced pain in both legs, more pronounced in the left limb. The leg pain was dull and aching in character, and not localized. The symptom [illegible]

C [illegible]

a te [illegible]

located in the middle of the sacrum. Routine laboratory and roentgen examinations were negative.

At operation on September 14, 1929, a yellowish soft friable tumor mass was found that protruded from the posterior surface of the sacrum and extended to its anterior surface; the area of bone erosion was approximately 2 cm. in diameter. A large amount of the tumor was removed by curettage. The pathologic diagnosis was chordoma. Following operation the patient was treated with radiation, receiving approximately 1,000 r to the sacrum.

On examination 6 months postoperatively, the patient still was free from pain, but a large mass was palpable in the rectum. Roentgenograms now revealed extensive destruction of the sacrum (Fig. 4). A second operation was performed, and the entire sacral canal was found to be filled with a fairly firm bluish-gray tumor mass that had displaced the sacral nerves laterally and appeared to incorporate them. It was deemed inadvisable, therefore, to attempt to remove this mass. Postoperatively, the patient received a second course of roentgen therapy of 1,000 r. The tumor decreased progressively in size and gradually became more firm. Four months after the second operation the patient was asymptomatic, and rectal examination revealed no increase in the size of the tumor. However, the symptoms subsequently recurred and became progressively more severe.

Approximately 3 years after the original operation, decompression and curettage of the sacral region again were performed. Immediately following operation the patient was relieved of much of the pain, but approximately 3 months later the pain recurred and the patient was given a third course of radiation therapy. Following this he experienced some alleviation of pain, but on rectal examination the size of the tumor was found definitely to have increased. A fourth operation that consisted of curettage of the tumor mass was performed approximately 7 years after the original operation. The patient died 2 months later, on December 13, 1936.

Just prior to death a tumor mass was noted in the jaw; this was biopsied and reported to be chordoma. At the time of death there was extensive recurrence at the site of the sacral operation, as well as at the site of biopsy in the mandible.

Vertebral chordomas exhibit a definite polarity, being primarily cervical or lumbar in location. Only a few cases have been reported in the thoracic region.[2,3,5] Our 6 vertebral chordomas were distributed as follows: 2 cervical lesions at C-2 and C-4, 3 lumbar tumors each located at L-3, and a solitary thoracic chordoma at T-11. In 2 instances, myelograms revealed spinal block.

Of all chordomas, those of the vertebrae probably present the most variable roentgen picture. Our 6 lesions all appeared to arise from the vertebrae. In each case the interspace was preserved on the initial examination, even when there was extension to an adjacent body or pedicle. We observed only limited involvement of the 2nd vertebra,

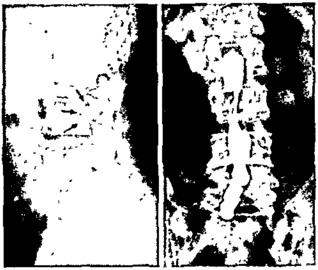


FIG. 5. (*Left*) Lateral roentgenogram of the lumbar spine shows a sharply demarcated osteolytic defect involving the posterior portion of body and pedicle of L-3. (*Right*) Myelogram demonstrates partial block at this level with narrowing of dye column.

with no extensive lesion. However, extensive destruction involving multiple vertebral bodies with narrowing of the interspace may occur.[13] All lesions in this series were osteolytic with a variable degree of bone involvement. Where changes were localized posteriorly to the region of the pedicle, the sharply demarcated erosion from slow growth suggested extrinsic pressure (Fig. 5). Differentiation from spinal tumor such as ependymoma, meningioma or neurofibroma may be impossible. In the cervical lesion at C-2, a soft tissue mass could be identified extending to the nasopharynx, but insufficient to cause obstruction of the air passage. This cervical lesion and the tumor involving D-11 were the only ones in our series that showed collapse of the vertebral body. Of 5 vertebral chordomas reported by Wood and Himadi,[15] all showed extensive involvement of 2 or more bodies with disk invasion and prominent extra-osseous extension. Unusual was the striking osteoblastic reaction in 2 of their cases. Increased bony density also has been observed by others.[3,12] Thus it would seem that neither reactive bone nor tumor calcification would allow the exclusion of chordoma. With lytic destruction of the entire body, differentiation from metastatic malignancy, as well as other primary neoplasms such as plasmocytoma, giant-cell tu-



FIG. 6. (*Left*) Left oblique roentgenogram of cervical spine with destruction of the lamina of C-4 and a portion of the lamina of C-3. Lytic destruction of the body of C-4 is demonstrated better on postoperative lateral view (*right*), which shows wiring of the spinous processes

mor, chondrosarcoma and aneurysmal bone cyst, rarely is possible. Infectious granuloma, particularly tuberculosis with an epidural component, may be similar in appearance to chordoma.

Case 4. This 56-year-old white female was seen first on May 26, 1955, complaining of pain in the neck of about 2 years' duration. The pain was aching and burning in character with extension around to both sides of the neck but no radiation to the shoulders, the arms or the hands. It was constant in character and had become progressively more severe. The physical findings were limited to the neck, where there was diffuse tenderness about C-3 and C-4, with restriction of neck motions in all directions, particularly extension.

Roentgenograms of the cervical spine showed osteolytic destruction of the 4th cervical vertebra and the pedicle on the left side at the level of C-3 and 4 (Fig. 6, *left*). Examination of the spinal fluid was normal.

A cervical laminectomy was performed on June 1, 1955, with removal of the spinous processes of C-2 and C-5. A portion of the lamina of C-4 on the left was removed as far as the pedicle. In this area there appeared a tumor mass, brownish-red in color and soft in consistency. Biopsy revealed this mass to be a chordoma. It was deemed inadvisable to remove the tumor. The procedure was completed with wiring of the spinous processes of C-3 to C-5 (Fig. 6, *right*). Bone chips were used for fusion. The patient was given cobalt 60 teletherapy, receiving approximately 6,000 r to the tumor.

The patient was seen last on August 10, 1955, at which time she had no significant pain.

Case 5. A 48-year-old white male was seen first on August 10, 1950, in Lorain, Ohio, because of pain in the left low back and flank that had begun 3 months previously. The pain did not radiate but had become progressively more severe. It was constant in character, severe enough to keep the patient awake at night, and was aggravated by stooping, bending, coughing and sneezing. The patient stated that before these symptoms developed he had been in good health except for a peptic ulcer.

The only abnormal finding on physical examination was tenderness to the left of the spines of L-2 and L-3 and in the left flank. Roentgen examination of the dorsal spine revealed concavity of the anterior border of D-11 with local destruction (Fig. 7, *left*). There was



FIG 7 (*Left*) Early destruction of 11th dorsal body limited to the anterior portion. (Doctors D A Russell & R. D. Berkebile, Lorain, Ohio) (*Right*) Later change with collapse of D-11 and involvement of anterior portion of D-10 but with interspace intact.

no collapse or evidence of bone sclerosis. The patient was considered to have vertebral tuberculosis. He was placed in a brace, and streptomycin therapy was begun.

Approximately 5 months later, roentgenograms revealed collapse of T-11 (Fig. 7, *right*) and a soft tissue mass in the region of the bony lesion that was thought to represent a tuberculous abscess. The patient continued to have severe pain. He developed drowsiness, listlessness and mental confusion, and was hospitalized at the Cleveland Clinic on January 30, 1951. The neurologic examination at that time was not remarkable, except for the mental confusion. On a 2nd spinal fluid examination the spinal fluid contained 69 mg. of protein, 2 white cells and 2,000 red cells, thought to be bloody tap. Spinal fluid was left to stand, and no pedicle formation was observed. The patient's mental condition subsequently improved, and he was discharged from the hospital but with an uncertain diagnosis.

Approximately 2 months later, some 11 months after onset of symptoms, the patient died. An autopsy was performed at St. Joseph's Hospital, in Lorain, which revealed that the lesion in the 11th dorsal vertebra was a chordoma, and that there were metastatic lesions in the lungs and in the lymph nodes of the neck.

Case 6. A 56-year-old white male was admitted to the hospital on the chief complaint of pain in the gluteal region that radiated down both legs to below the knees. The pain had begun approximately 1 year previously, but had become extremely severe during the 2 months preceding admission. The pain was aching in character and radiated posteriorly down both legs laterally and anteriorly with aggravation on lying down. Three years previously the patient had a nephrectomy for hydronephrosis.

On admission, physical examination failed to reveal any objective musculoskeletal abnormality, and the neurologic examination was essentially negative. On routine laboratory examination, 50 mg. of protein was found in the spinal fluid Roentgenograms of the lumbar spine showed an osteolytic area of destruction of the 3rd lumbar vertebra, localized in the posterior portion of the body and the pedicle. A myelogram showed a large extradural defect at this level (Fig. 5).

At operation on February 11, 1954, the laminae of L-2 and L-3 were removed An apparently encapsulated chordoma was found in the posterior part of the body of L-3, compressing the 4th lumbar nerve. The tumor was incompletely removed.

Postoperatively, the patient was given a course of radiation therapy, receiving 3,000 r to L-3. He was discharged from the hospital free from pain, but approximately 1 year later pain recurred in the left buttock, thigh and knee. Roentgen examination at that time showed no marked change from the previous examinations. The pain continued, and, at last follow-up, approximately 1 year postoperatively, the patient was working and taking analgesics for relief of pain.

PROGNOSIS AND TREATMENT

Clinically, metastases are uncommon. They are reported as occurring primarily from tumors in the sacrococcygeal region. In Mabrey's series[9] of 16 autopsied caudal lesions, 10 showed metastases, an incidence of 62 per cent. The most common sites were the regional lymph nodes, with liver, pleura and peritoneum being less frequently involved. Isolated instances of metastases from chordomas in other locations have been reported,[10,11] some with widespread involvement. Metastases occurred in 2 patients in our group (Cases 3 and 5); in 1, the metastasis was to the mandible from a sacrococcygeal tumor, and in the other it was to the lungs and the neck from a dorsal tumor.

Curative treatment of chordoma can be achieved only by total surgical removal of the tumor. This, however, is almost invariably impossible because of the nature and the location of these growths. Removal is limited by inaccessibility and the presence of vital structures in the cranial and the vertebral locations. It is only in the sacrococcygeal location that adequate excision is feasible, and then only rarely. In none of our 19 patients could radical excision be attempted. In the vertebral location, biopsy with laminectomy for relief of pressure was the usual procedure. When a large tumor mass is present, removal of as much of the mass as possible affords considerable relief for a variable period of time. Recurrence is the rule, and repeated operations may be necessary for symptomatic relief. Few, if any,

of the patients with chordoma ever are cured. Death usually is due to local extension.

Radiation is only of palliative value, since most of these tumors are radioresistant. Temporary tumor regression may be observed, affording considerable symptomatic relief. Following radiation, Poppen and King[12] have reported bone formation and disappearance of a tumor at reoperation. Sterilization of the growth, however, rarely is possible, except in the extremely unusual radiosensitive lesion. Tumor dose has varied from 1,000 r with conventional voltage to 6,000 r given with cobalt teletherapy in 2 of our vertebral tumors. Sufficient time has not elapsed in these 2 cases for evaluation, but it is doubtful that the long-range result can be altered significantly by any form of radiation.

In the evaluation of the results of therapy, the usual 5-year cure rate is of little value because of the protracted course of this tumor. The longest survival in our group, a patient with a sacrococcygeal lesion, is 18 years; the patient still is alive but has a recurrence. Dahlin and MacCarty[3] described a patient who survived 23 years with a sacrococcygeal tumor. Six of the 9 patients with cranial neoplasms in our series were followed until they died. The average time that elapsed from initial surgical treatment until death was 44 months, with 2 patients each living 9 years postoperatively. Three were alive 2½, 5 and 5½ years postoperatively, but 2 of the 3 had recurrence.

As a rule, sacrococcygeal lesions tend to be less rapidly fatal than cranial lesions. Increased survival is expected because of less vital adjacent structures and greater freedom for surgical intervention. Of our 4 patients with sacral chordomas, 1 died 7 years after surgery, and the other 3 were all living at last follow-up, averaging 7 years postoperatively. All had known recurrence.

Because treatment of our vertebral group was administered so recently, no adequate survival data are yet available. The patient with the lesion at D-11 died 11 months after initial onset of symptoms. The remaining 5 patients were all alive when last heard of, none free of disease.

SUMMARY

Chordomas are rare neoplasms which arise from notochordal remnants along the cerebrospinal axis; they occur most commonly at the extremes of this axis. Growth is slow, with extension of the tumor to adjacent structures. Symptoms can be of long duration, and they vary with the site of origin.

Clinical and radiologic findings in the cranial, the sacrococcygeal and the vertebral locations are discussed, and representative case reports presented.

Treatment is surgical, with radiation of some palliative value. Almost invariably, total removal is impossible, and recurrence is the rule. Metastases are uncommon. Death usually results from local extension after a prolonged postoperative course.

REFERENCES

1. Arey, L. B.: Developmental Anatomy, ed. 6, Philadelphia, Saunders, 1954.
2. Crowe, G. G., and Muldoon, P. B. L.: Thoracic chordoma, Thorax 6:403, 1951.
3. Dahlin, D. C., and MacCarty, C. S.: Chordoma; a study of 59 cases, Cancer 5:1170, 1952.
4. Dickson, J. A., and Lamb, C. A.: Sacral chordoma, Ann. Surg. 93:857, 1931.
5. Faust, D. B., Gilmore, H. R., Jr., and Mudgett, C. S.: Chordomata: review of literature with report of a sacrococcygeal case, Ann. Int. Med. 21:678, 1944.
6. Freeman, J.: Cranial chordoma, Arch. Otolaryng. 51:237, 1950.
7. Freeth, A., and Mair, J.: Chordoma as cause of obstetric disproportion, Brit. M. J. 1:512, 1951.
8. Gardner, W. J., and Turner, O.: Cranial chordomas, a clinical and *pathologic study*, Arch. Surg. 42:411, 1941.
9. Mabrey, R. E.: Chordoma: study of 150 cases, Am. J. Cancer 25:501, 1935.
10. Morris, A. A., and [illegible]
11. Pastore, P. N., Sahyoun, P. F., and Mandeville, F. B.: Chordoma of the *maxillary* antrum and nares, report of case clinically

resembling Hodgkin's disease first diagnosed by biopsy of cervical node, Arch. Otolaryng. 50:647, 1949.
12. Poppen, J. L., and King, A. B.: Chordoma: experience with 13 cases, J. Neurosurg. 9:139, 1952.
13. Roaf, R.: Chordoma of the third lumbar vertebra, J. Bone & Joint Surg. 32-B:40, 1950.
14. Williams, L. W.: Later development of the noto-chord in mammals, Am. J. Anat. 8:251, 1908.
15. Wood, E. H., Jr., and Himadi, G. M.: Chordomas: a roentgenologic study of 16 cases previously unreported, Radiology 54: 706, 1950.

Chordoma

Summario in Interlingua

Chordomas es rar neoplasmas de basse malignitate, originari del proliferation de restos notochordal in cranio e columna vertebral Illos es situate le plus frequentemente in cranio e region sacrococcygee. Etiam le vertebras pote esser afficite, usualmente in le region lumbar e cervical.

Signos e symptomas depende del sito. Tumores cranial occurre in le region del clivo, sed illos pote extender se considerabilemente con le effecto de definite indicios neurologic pro tumores intracranial Le constatationes roentgenologic es indicative de destruction ossee; in certe casos on nota le presentia de un massa calcificate.

In tumores sacrococcygee le symptoma ducente al consultation medical es usualmente dolores que pote esser associate con un massa de texito molle. Le configuration roentgenologic revela un irregular defecto osteolytic in le area central.

Tumores vertebral exhibi un predilection pro le region cervical e lumbar. Illos affice le corpore o le pediculo del vertebra e a vices se extende a vertebras vicin. Sclerosis ossee, collapso del corpore, e restriction del interspatio pote occurrer.

Ab le puncto de vista clinic il es a signalar que metastases es infrequente. Inter le varie typos de chordoma, illos del region sacrococcygee resulta le plus frequentemente in metastases, usualmente a nodos regional. Extensissime disseminationes ab chordomas sacrococcygee e vertebral es de occurrentia rar.

Le therapia es relativemente dissatisfacente. Proque le tumores occurre in le vicinitate de tante structuras vital, le complete ablation chirurgic es quasi impossibile, excepte in le caso de favorabilemente situate lesiones sacrococcygee. Le crescentia del tumores es lente. Recurrentias es le norma de maniera que repetite interventiones chirurgic deveni necessari. Le curso clinic es de longe duration. Radiation pote resultar in regression e alleviamento del dolar, sed illo non es curative. Le morte resulta usualmente ab extension local.

Es presentate typic historias de casos de chordoma in varie sitos, insimul con pertinente radiogrammas.

11

Osteoid Osteoma; Report of Atypical Cases

WILLIAM R. DAVISON, M.D.*

Osteoid osteoma now is recognized as a definite pathologic entity, and in most instances its diagnosis is readily made by the history of the case and roentgenologic features.

When the lesion is in the vicinity of joints, symptoms of joint involvement may be present; however, roentgenologic study usually reveals the lesion. Although formation of reactive bone is a constant feature when the lesion is located in cortical bone or occupies a subperiosteal position, this feature may be absent if the lesion is in cancellous bone. Nevertheless, the nidus is discernible in roentgenograms. However, cases are encountered occasionally in which the nidus is not demonstrable, particularly when the lesion occupies a subchondral position. In these instances, diagnosis is almost impossible before operation. Such lesions may present bizarre and misleading clinical features. Two such cases are presented in this report.

Occasionally, only the clinical manifestations may permit a presumptive diagnosis of osteoid osteoma. Such a case also is reported herein, in which roentgenograms and laminagraphs failed to disclose the nidus. The lesion was demonstrated at the time of surgery.

Recurrence of a lesion after removal or the appearance of a second lesion is indeed rare, yet in 1 case the sequence of events leads us to believe that this is possible.

Finally, a case is reported of a female aged 72. The oldest case reported in the literature is 60.

For the sake of completeness, the usual clinical features of osteoid osteoma will be reviewed.

* Philadelphia, Pa.

CLINICAL CONSIDERATIONS

AGE

Reports vary as to the greatest age incidence. Some say that it is from 10 to 20 years; others, from 5 to 15 years. Suffice it to say that it has a predilection for the adolescent and the young adult. The youngest case was 14 months of age; the oldest, 60 years of age.

SEX

In most series the male predominates, the ratio ranging from 2 to 1 to 4 to 1 and higher.

LOCATION OF LESIONS

The lesion has been reported in every bone with the exception of the clavicle, the scapula and the skull. The long bones of the lower limbs are the sites most frequently encountered, the tibia being the most frequent and the femur next. One case has been described as occurring in the femoral epiphysis; 2 cases have been reported in which 2 lesions occurred in 1 bone.

SYMPTOMS

Clinically, pain is the most outstanding feature of this lesion, and it may precede by many months any development of roentgen abnormalities. In rare cases, spontaneous pain may not be the presenting complaint. Pines, Lavine and Grozzed reported 2 cases in which the chief complaint was limping. The duration of symptoms has been reported as ranging from 2 weeks to 35 years. Jaffe states that histories of less than 6 months are

the exception; of 6 months to 2 years the rule; and of over 2 years uncommon. The pain is mild and intermittent at first, but usually it increases in persistence and severity, and frequently awakes the patient at night. When the lower limb is involved, a limp frequently develops. The striking feature of this entity in its early stage is the local pain with few physical and roentgenographic findings to support it. The pain is aggravated frequently by exercise and relieved by rest and 10 grains of aspirin. When the lesion is found in the spine, there may be associated root pain, or even symptoms of cord compression.

On physical examination, a sharply demarked point of tenderness is found somewhere within the painful area. If the lesion is superficial, there may be some slight swelling, and, on occasion, the area takes on the appearance of inflammation, although this is rare. Adjacent joints may be swollen with limitation of motion, as some patients give a history of intermittent joint swelling and stiffness. Weakness and muscle atrophy may be seen in some cases in which the part has been protected One patient with a lesion in the posterior portion of a rib developed a secondary scoliosis, compensatory lesion that is seen also in osteoid osteomas involving the spine. Kleinburg suggested the clinical triad of (1) insidious but continuing discomfort, (2) localized pain and (3) localized tenderness.

Very few cases have been encountered in which the patients have had a generalized reaction such as fever and leukocytosis, and some authors state that the above findings should cast doubt on the diagnosis.

Roentgenographic Findings

X-ray is the most valuable diagnostic aid, the nidus being pathognomonic of the entity.

The nidus appears as a small oval circumscribed radiolucent, or less frequently radiopaque, area measuring from a few millimeters up to 2 cm The nidus is surrounded invariably by a zone of sclerotic bone that varies in width. It is this reactive bone that surrounds the nidus which at times blocks out the nidus under ordinary conditions of exposure. In cancellous bone, the surrounding sclerosis usually is less than that seen when the lesion is near the cortex.

While, originally, Jaffe and others believed the radiolucency of the nidus to be an early stage in the evaluation of the tumor and radiopacity to be a sign of maturity, he has since stated that he believes the reverse to be nearer the fact. Hence, the difficulty in diagnosing an early case may stem from the fact that the surrounding sclerosis blends with that of the nidus. On the other hand, if the lesion is seen early enough and the surrounding bone has not undergone much reaction, the nidus may stand out as a focus of increased density.

When the lesion is found in proximity to joints, degenerative changes sometimes can be demonstrated.

Pathologic Findings

Jaffe has described this lesion as a benign osteogenic tumor of slow growth, with the initial phase of evaluation being one of proliferation of local bone forming mesenchyme and particularly osteoblasts. Under the microscope, the lesion consists of a substratum of vascular, richly cellular embryonal osteogenic connective tissue. Within this connective tissue are seen variable amounts of calcified and uncalcified osteoid tissue, and trabeculae of newly formed atypical bone and numerous giant cells. Osteoblasts usually are abundant, and osteoclasts are scarce. While in some the osteoblasts and other mesenchymal components have a normal appearance, in others the cells are large, irregular and very numerous with hyperchromic nuclei showing some evidence of mitosis. The amount of surrounding osteosclerosis depends on the location of the nidus. When it arises in the spongiosa, it frequently initiates a perifocal osteosclerosis of greater or lesser extent, although this is not constant. When the nidus is located in the cortex, the surrounding sclerosis is invariably greater

in its extent and often incites the periosteum to formation of great deposition of new bone formation. Occasionally the lesion is limited to periosteum, and here the reaction is minimal.

Pathogenesis

The exact etiology of this lesion has yet to be fully agreed upon. Jaffe, Lichtenstein and others hold to their theory that the lesion is a benign tumor of bone, while others find cause to believe that inflammation plays a role. In isolated cases, organisms have been demonstrated in these lesions, but certainly not consistently enough to bear out the hypothesis that inflammation plays a role. Inflammatory cells are consistently absent from the pathologic picture. The only site at which inflammation has been observed in these lesions is in adjacent joints, where a synovitis may exist. Some contend that the lesion is an embryonal arrest, while others believe it to be a healing process resulting from trauma, but so far these theories have not been substantiated.

Treatment

The prime objective in the treatment of this lesion is removal of the nidus. The technic for this procedure varies; some merely curet the area, while others believe that block resection is the method of choice. The difficulty with either is the chance of missing the nidus. The only disadvantage with curettement is that the lesion usually is crumbled, hence it is difficult to pick out the exact fragment that will give the histologic diagnosis.

Some unusual clinical and roentgenographic findings are shown in the following cases:

CASE HISTORIES

Case 1. A 10-year-old boy presented himself in December, 1953, because of pain in the left knee and shortness of the left leg. At the age of 3 the boy complained frequently of pain

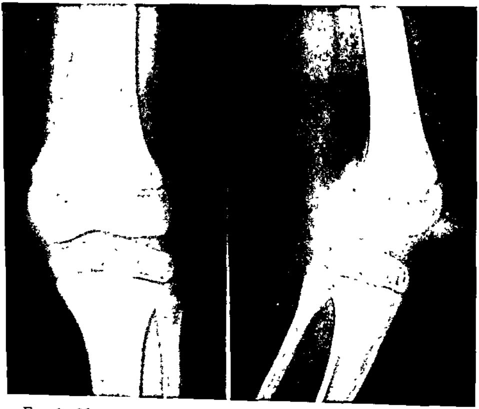


Fig 1. No evidence of the involvement of the inferior pole of the patella is demonstrable. The only roentgenographic finding is some decreased density of the bone as a whole.

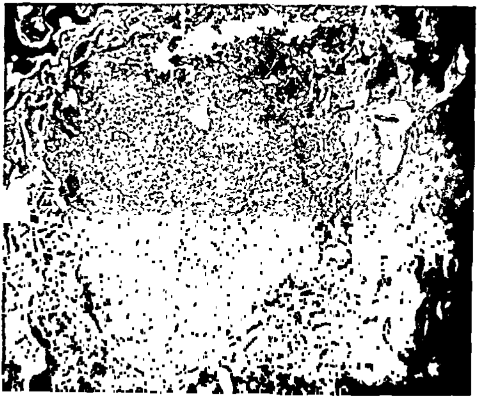


FIG. 2. Tissue removed from the inferior pole of the patella shown in Figure 1. It is consistent with a typical case of osteoid osteoma.



FIG. 3. Note the sclerotic bony region implicating the distal third of the femur. The entire femur has been stimulated to increased growth, so that it is slightly longer than the one on the opposite side. Roentgenographic features are consistent with an osteoid osteoma.



FIG. 4. Tissue obtained from the specimen removed in the case illustrated in Figure 3. It is consistent with an osteoid osteoma.

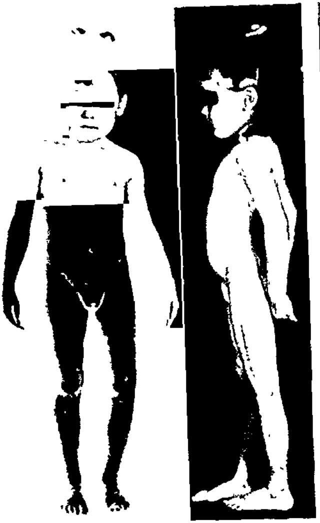


FIG. 5. Following surgery, note that the child has developed a varus deformity of the left extremity. At this time the patient again developed severe pain in the lower region of the outer aspect of the femur.

throughout his left lower extremity. This complaint persisted for from 2 to 3 years, at which time he began to limp, and it was noticed that the left leg was smaller than the right.

Physical examination disclosed atrophy of the thigh and the calf muscles with associated loss of power. There was exquisite tenderness overlying the area of the patella.

All laboratory studies were within the limits of normal.

Roentgenographic studies of the left knee showed some osteoporosis of the patella. Also, the bones of the left knee joint region showed generalized osteoporosis. (Fig. 1.)

Because no lesion could be demonstrated and the symptoms failed to respond to conservative therapy, the area was explored. A small bleb was found on the lower pole of the patella, and this area was resected. Upon sectioning this

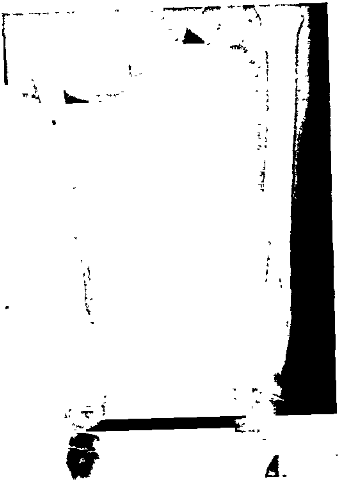


FIG. 6. Note the lesion on the outer aspect of the lower end of the left femur, which is consistent with an osteoid osteoma.

specimen, a nidus was found. (Fig. 2.) Immediately after operation the patient was relieved of his pain.

This case is difficult from a diagnostic viewpoint. The absence of reactive bone surrounding the nidus makes it a rarity.

Case 2. A 4-year-old girl was admitted to the hospital in May, 1950, with the history that since she first walked she had favored the left leg. Four months prior to admission she first complained of a vague pain in the thigh, which at times awakened her at night.

Physical examination revealed a bony enlargement of the lower third of the femur which was tender to palpation.

A roentgenogram showed marked hyperostosis of bone, apparently produced by periosteal new bone formations along the lateral aspect of the femur in the distal half. (Fig. 3.) The radiologists ventured a diagnosis of infantile cortical hyperostosis.

At operation, a piece of the shaft of the femur, measuring more than one half of the circumference and 6½ cm. long, was removed.

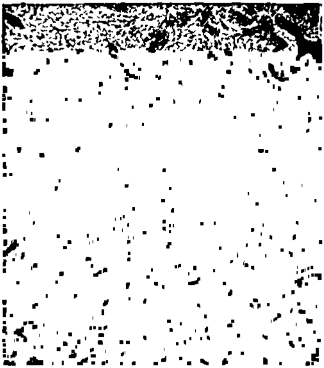


FIG 7. Tissue removed from the specimen obtained at the site of the lesion shown in Figure 6.



FIG. 9. Tissue consistent with osteoid osteoma removed from the case shown in Figure 8.



FIG 8 *(Left)* The arrow points to a small area of increased density in the cortex of the outer aspect of the femur. *(Right)* A block resection has been done of the area depicted.



FIG. 10 Note the increased density and thickening of the base of the shaft of the 5th metacarpal bone. The entire area was resected as disclosed in Figure 11.

A fusiform area of pale pinkish-gray granular material was found just beneath the cortex. This was taken to be the nidus. Histologic examination confirmed this finding. (Fig. 4.)

The child was asymptomatic immediately after operation, and remained so until October, 1952, when she complained of a dull pain at the lower pole of the previous incision. This area was extremely tender. There was a varus deformity of the leg. (Fig 5.)

A roentgenogram showed an area of sclerosis extending beneath the cortex, and the lateral condyle was ¼ in. longer than the medial one. (Fig. 6.) At operation, a block incision was made of the involved segment, and grossly this appeared to be porous and hemorrhagic. Histologic examination revealed the typical findings of an osteoid osteoma. (Fig. 7.)

The literature cites numerous cases of recurrence following incomplete removal of the nidus, but these patients were not relieved of their pain. This patient was asymptomatic for 2 years following the first procedure. In reviewing her slides, it was felt the entire nidus was removed at the first operation. It would appear that this was a new lesion.

Case 3. A 73-year-old female was seen first in December, 1952, complaining of pain in her right thigh that had been constant since the onset in 1950. It was aggravated by exercise and relieved by rest. Two months prior to her admission to hospital, the pain no longer was relieved by rest and was increasing in severity. There was no history of trauma.

Physical examination revealed a firm swelling just distal to the greater trochanter on the right thigh which was tender. Considerable muscle spasm was present.

Roentgenograms of the right femur revealed a radiopacity in the cortex of the upper third of the femur. The preoperative diagnosis was osteoid osteoma. (Fig. 8, *left.*)

At operation, a section of the involved area

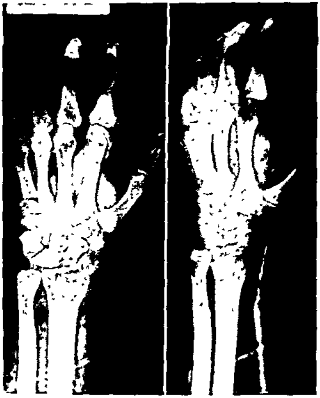


FIG. 11. The entire portion of the base of the 5th metacarpal shaft was resected. Histologic examination of this tissue disclosed a typical case of osteoid osteoma.

was removed in block, and on sectioning this a nidus was found. (Fig. 8, *right.*)

The patient's postoperative course was complicated by a fracture through the operative site, sustained when she fell 2 weeks after surgery. (Fig. 9.) This was treated by open reduction and intramedullary fixation. The patient has had no recurrence of symptoms to date

In the light of our present knowledge of this lesion, the age of this patient is unusual. Sixty was the most advanced age reported, by Dahlin and Johnson in their series of giant osteoid osteomas.

Case 4. A 33-year-old male complained of pain in his left hand that had persisted for 2 years prior to his admission to the hospital in July, 1951. He slammed a drawer on his hand at that time, and subsequently developed pain and inability to move the 4th and the 5th fingers. This became progressively worse until July, 1952, when this area was explored at another institution without relief of symptoms. The severity of the pain reached such a degree *that he requested amputation on admission*

Physical examination r ealed atrophy of the hypothenar prominence o the left The fingers appeared shiny and red th decreased motion. There was tenderness over the 4th and the 5th metacarpal phalangeal joints.

A roentgenogram revealed in the region of the base of the 5th metacarpal bone an area of increased density, with some slight expansion of the bone. (Fig. 10.)

At operation, the periosteum in this area was thickened with an erosion of the cortex. Degenerative changes of the adjacent joints was present in this area. Because of this the proximal one third of the 5th metacarpal was resected. (Fig. 11.)

The patient experienced complete relief of symptoms.

Case 5. A 21-year-old male was seen first in August, 1948, complaining of pain and swelling in the area of the 2nd metacarpal phalangeal joint of the left hand. He first noticed the pain in May, 1947. Prior to this admission the patient had had 2 operative procedures for the same complaint. The first procedure was an incision and drainage, and the second was for removal of a neurofibroma. The pain became much more severe and kept the patient awake at night He had a lower cervical sympathetic block without relief. This was followed by immobilization in a cast for 7 weeks and later roentgen therapy.

Physical examination revealed a tender mass over the metacarpophalangeal joint of the 2nd finger. There was limitation of motion of the index finger. The hand was sweaty to the touch and had a red, mottled appearance

The diagnosis at this time was a tenosynovitis, and the flexor tendon sheath was excised, along with the scar tissue surrounding the nerve.

The patient was readmitted to the hospital in October, 1948, with essentially the same complaints and findings. At this time the diagnosis was a recurrent neurofibroma, and the digital nerve was resected.

The patient was seen again in January, 1949. The complaints were similar, but now his finger was very swollen. At this time various consultants believed the process to be either an infectious lesion or a neurofibroma.

Roentgenograms of the finger and involved joints were read as negative on both previous admissions and also the present admission (Fig. 12.)

Because the patient requested amputation and no other alternative could be decided upon, this was performed. On resectioning the bones a soft grayish-pink *granular lesion* was found in the base of the proximal phalanx. (Fig. 13.) Histologically this was an osteoid osteoma (Fig. 14.)

Cases 4 and 5 are similar in their clinical history and course, yet they vary greatly in the roentgenographic appearance. The former case revealed a small nidus of increased density and the absence of reactive bone surrounding the lesion. Because of the severity of the reaction in the adjacent joints, it was felt that a resection of the involved portion of bone would prevent a painful arthritis from developing in this area. The latter case was extremely interesting and also unfortunate in that the patient had to lose his finger before a diagnosis was made. It is difficult to account for the essentially

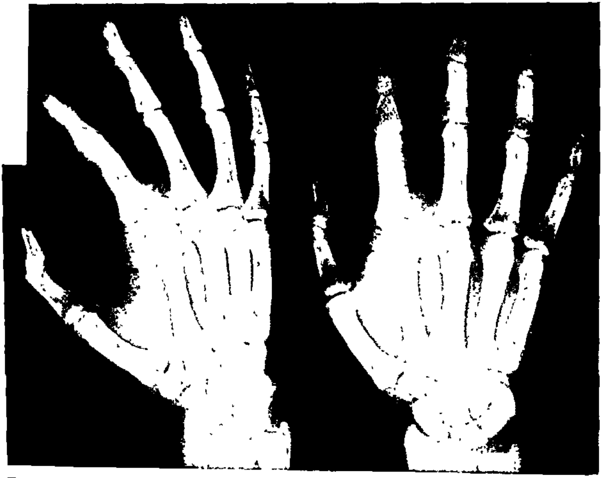


FIG. 12. Except for some decreased density of the 2nd metacarpal bone, there is no evidence of any bony pathology.



FIG. 13. This is the postoperative specimen obtained after amputation of the index finger of the case shown in Figure 12. *Note the nidus in the base of the proximal phalanx.*

normal appearance of the skeleton in the area of this lesion, and this explains why amputation was performed. That biopsy would have benefited in this case has been proved by the final diagnosis, but evidence as to the exact site to biopsy was lacking. No case with similar findings or absence of findings can be found in the literature.

We have presented 5 cases of osteoid osteoma showing some unusual aspects both clinically and roentgenographically. Two cases have been presented that failed to show any nidus or reactive bone on roentgenogram. It would seem that in similar cases in which the clinical picture is typical of that seen in this lesion, an exploratory procedure should be carried out.

BIBLIOGRAPHY

Brailsford, J. F.: Brodie's abscess and its differential diagnosis, Brit. M. J. 2:119, 1938.

———: Chronic sub-periosteal abscess, Brit J. Radiol. 15:313, 1942.

Brown, R. C., and Ghormley, R. K.: Solitary eccentric (cortical) abscess in bone, Surgery 14:541-553, 1943.

Dahlin, David C., and Johnson, E. W., Jr.: Giant osteoid osteoma, J. Bone & Joint Surg. 36-A:559, 1954.

Dockerty, M. B., Ghormley, R. K., and Jackson, A. E.: Osteoid osteoma; a clinicopathologic study of 20 cases, Ann. Surg. 133:77-89, 1951.

Jaffe, H. L.: Osteoid osteoma; a benign osteoblastic tumor composed of osteoid and atypical bone, Arch. Surg. 31:709-728, 1935.

———: Osteoid osteoma of bone, Radiology 45:319-334, 1945.

Jaffe, H. L., and Lichtenstein, L.: Osteoid osteoma; further experience with this benign tumor of bone with special reference to cases showing lesions in relation to shaft cortices and commonly misclassified as instances of sclerosing non-suppurative osteo-myelitis or cortical bone abscess, J. Bone & Joint Surg 22:645, 1940.

Kleinberg, S.: Osteoid osteoma of the femur; report of a case, Am. J. Surg. 53:168, 1941.

———: Osteoid osteoma; report of five cases, New York J. Med. 43:332-338, 1943.



FIG. 14 Typical tissue of osteoid osteoma obtained from the nidus shown in Figure 13.

Lewis, R. W.: Osteoid osteoma; a review of portions of the literature and presentation of cases, Am. J. Roentgenol. 52:70, 1944.
Mallory, T.: Case report of Massachusetts General Hospital, New England J. Med. 225: 920, 1941.
Ponseti, I., and Barta, C. K.: Osteoid osteoma, J. Bone & Joint Surg. 29:767, 1947.
Sherman, M. S.: Osteoid osteoma associated with changes in adjacent joint; report of two cases, J. Bone & Joint Surg. 29:485-490, 1947.
———:Osteoid osteoma; review of the literature and report of thirty cases, J. Bone & Joint Surg. 29:918-930, 1947.

Osteoma Osteoide; Reporto de Casos Atypic

Summario in Interlingua

Osteoma osteoide es hodie recognoscite como un difinite entitate pathologic. Su diagnose se face in le majoritate del casos facilemente super le base del historia del patiente e del aspectos del roentgenogramma. In le presente articulo nos passa in revista cinque casos que exhibí certe aspectos inusual de osteoma osteoide. Duo del casos esseva simile in que illos evocava nulle pathologia ossee in le roentgenogramma. Le prime caso concerne un puero de dece annos qui habeva suffrite dolores del genu deposit septe annos. Le dolores esseva associate con claudication. Al operation un osteoma osteoide esseva trovate al polo inferior del patella. Le secunde caso es illo de un masculo de 21 annos qui se plangeva de dolores al secunde articulation metacarpo-phalangee del mano sinistre. Le dolores habeva durate circa duo annos. Iste patiente habeva habite tres previe operationes pro varie diagnoses del area in question. Proque nulle diagnose poteva esser establite, un amputation esseva executate. Post sectionar le ossos del digito, un osteoma osteoide esseva trovate al base del phalange proxime. Iste caso suggere que in simile situationes, quando le aspectos clinic es del typo hic observate e quando le roentgenogramma es negative pro pathologia ossee, un operation exploratori deberea esser executate

Proque le majoritate del reportos in le litteratura indica que iste entitate es characterisate per su predilection pro adolescentes e juvene adultos, nos crede interessante reportar hic le caso de un femina de 73 annos qui habeva un lesion in le extremitate proxime del femore dextere. Le patiente del etate le plus avantiate usque nunc reportate es un patiente de 60 annos reportate per Dahlin e Johnson.

In multe reportos recurrentias de iste lesion es mentionate, sed in le majoritate del casos tal recurrentias esseva debite al inadequate ablation executate al tempore del prime operation. Le difficultates technic incontrate in determinar le exacte location del nevo es ben cognoscite, sed nos presenta hic un caso in que nos opina que le nevo esseva integremente abferite al tempore del operation. Pro confirmar iste opinion, nos possede le protocollo del examine histologic que indica que le nevo esseva vermente eliminate e le constatation clinic que le patiente esseva completemente asymptomatic immediatemente post le operation. De facto, le patiente remaneva asymptomatic durante circa duo e medie annos. Post iste intervallo le patiente se re-presentava a causa de dolores in le mesme loco. Nostre diagnose a iste tempore esseva recurrentia de osteoma osteoide, e isto esseva de novo confirmate al operation e per sectiones histologic.

Le ultime caso concerne un masculo de 31 annos qui habeva suffrite dolores del mano sinistre durante duo annos ante su admission al hospital. Le roentgenogramma revelava in le region del base del quinte osso metacarpal un area de augmentate densitate con alterationes degenerative presente in le articulation adjacente. A causa del affection del articulationes in iste area, nos credeva necessari executar un resection del tertio proxime del quinte osso metacarpal. Le operation resultava in le complete alleviamento del symptomas pro le patiente. Nos considera iste caso como interessante a causa del affection articulari observate in illo e a causa del methodo de tractamento que resultava in le complete alleviation del symptomas.

12

Synovial Chondromatosis; a Report of Two Cases

H. KELIKIAN,* M.D., AND SHERMAN S. COLEMAN,* M.D.

Synovial chondromatosis and synovial osteochondromatosis, though separable both clinically and pathologically, are variations of the same disease process. The condition affects the synovial membranes of joints and bursae; and it has also been reported to occur in tendon sheaths. In each case its characteristic pathologic feature is a large number of intramural or intra-articular bodies composed of hyaline cartilage and/or bone. Numerous cases of synovial *osteochondromatosis* have been described; but reports of *chondromatosis* of the synovial membranes are far less common. It is our purpose to report here 2 cases of synovial chondromatosis and to emphasize some of the problems in diagnosis.

ETIOLOGY

The cause is unknown. Proposed theories include neoplasia, trauma, infection, embryonic cells "rests" and metaplasia None has been proved, and none has received general acceptance. It has been suggested that the course of the disease may be self-limited;[9,11] on the other hand, the development of chondrosarcoma has been observed in some.[3,8] Notwithstanding the obscure nature of the disease, it is apparent that the principal disturbance is an abnormal proliferation of cartilage within the stratum synoviale of synovial lined cavities.[7] Since cartilage cells may exist in small numbers in the synovial villi of normal joints,[6] it is reasonable to suspect that this capacity to form cartilage may be a holdover from the primitive mesenchymal origin of the synovial membrane and capsule.

PATHOLOGY

The disease is characteristically monoarticular. The knee is affected most often, followed in frequency by the elbow, the hip, the wrist and the ankle.[9] The synovial lining is thickened, and the joint cavity is packed with cartilaginous masses of varying size which may number in the hundreds These may be free, attached to the synovial membrane by a vascular pedicle, or embedded within the superficial layers of the synovial membrane.

The synovial lining often shows varying degrees of low grade chronic proliferative inflammation. In some of the more cellular areas, the cells in the synovial membrane appear to transform into cartilage cells. The greatest production of the nests of cartilage usually occurs at the point of reflection of the synovial membrane from the capsule onto the articular cartilage.[5] Interestingly enough, this corresponds to the transitional zone, where, in the developing joint, the cells possess the potentiality to form either hyaline cartilage or synovial tissue.[10]

The method of formation of cartilage and bone within the synovial membrane recapitulates the histogenesis of cartilage as observed in the embryo.[4,12] Thus, areas of loose vascular connective tissue blend with mesen-

* Chicago, Ill

chymal tissue, which in turn can be traced into cartilage formation. In synovial *chondromatosis* the process ceases at this point. None of the cartilage masses become either calcified or ossified; rather, they retain their purely cartilaginous composition. On the other hand, in the case of synovial *osteochondromatosis,* this process proceeds through the stage of calcification, culminating in osteoid or true bone formation. In such cases the osteochondral bodies exhibit either calcification or ossification within their central portions. In the former, hyaline cartilage is calcified, but there is no bone formation. In the latter, the centers of the bodies are composed of true bone. It has been suggested that calcification may occur either in loose or attached cartilaginous bodies, whereas bone can form only in those which retain a vascular pedicle.[2] This is biologically sound, but difficult to prove.

CLINICAL AND RADIOLOGIC FINDINGS

Whether one is dealing with a case of synovial *chondromatosis* or *osteochondromatosis,* the symptoms are much the same. These are pain, swelling and limitation of motion of the involved joint. Usually the symptoms begin insidiously and increase gradually over a period of months, or even years. Sometimes a history of "locking" can be elicited, and occasionally the first symptom the patient notices is the presence of a movable mass within the joint. The clinical signs include enlargement of the joint, which is perceived as a boggy synovial thickening rather than as increased intra-articular fluid. Crepitation and pain on passive motion are common; and nearly always there is a demonstrable limitation of joint motion.

The roentgenographic picture varies, de-

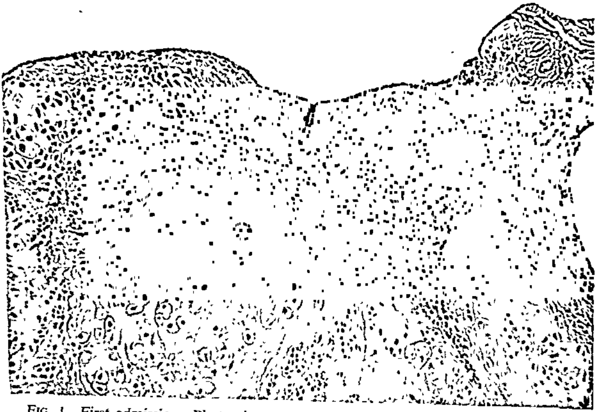


FIG. 1. First admission. Photomicrograph of section, depicting the nests of cartilage cells that have developed within the synovial membrane. Areas of normal synovial tissue can be seen occasionally in the section × 80.



FIG. 2. Second admission. Roentgenogram of elbow, showing absence of any significant changes in the bones. There is a suggestive fullness of the antecubital space, but this is nonspecific.



FIG. 3. Second admission. Pneumarthrogram of elbow, showing air dispersed throughout a large distended joint cavity filled with irregular masses having about the same density as the surrounding soft tissue.

pending upon whether the loose bodies are calcified or ossified, or whether or not they are purely of cartilaginous composition. In the former, the masses are seen clearly as round or oval structures of increased density, being intra-articular in location and possessing either a mottled or a definitely osseous appearance. If the masses are exclusively cartilage, usually the plain films are interpreted as negative; or merely a soft tissue swelling is seen which may or may not appear to be intracapsular. In these cases the diagnosis of synovial chondromatosis can only be surmised, and usually the condition is clinically misdiagnosed as either rheumatoid[1] or tuberculous[7] arthritis. Pneumarthrography can be employed as an aid in corroborating the diagnosis, as illustrated in the following reports.

Case 1. A 47-year-old white female was admitted to the hospital on March 15, 1948, complaining of a slowly progressive swelling and limitation of motion of the left elbow. She first became aware of the trouble about 5 years before, when she noticed difficulty in combing her hair. Subsequently she developed pain, which later became severe enough to keep her awake at night.

Examination on admission was essentially negative, except for the left elbow, which obviously was enlarged. There was a boggy nonfluctuant swelling on palpation, most marked over the antecubital fossa. The enlargement was firm but compressible. Joint motion was limited to a range of 65° (135-70). There was pain on pressure or on active or passive motion. The roentgenogram was interpreted as essentially normal

Arthrotomy was performed on March 20, 1948. The elbow was opened through a posterior incision, and numerous loose bodies of cartilaginous consistency were found. They measured up to 1½ cm. in diameter. A synovectomy was done. The postoperative course was uneventful, and a satisfactory range of painless joint motion was regained within a few months. The pathologic diagnosis was synovial chondromatosis (Fig. 1).

On August 26, 1953, the patient was readmitted to the hospital. The left elbow had been asymptomatic following surgery until 1 year prior to the second admission, when she noted a gradually developing painless swelling of the upper one third of the left forearm.

Examination again revealed a compressible fulness of the antecubital fossa and the upper end of the left forearm. Plain films of the elbow (Fig. 2) were reported as negative for any disease of the bones or the joint. A pneumarthrogram showed definite space-occupying lesions of the elbow joint (Fig. 3).

Arthrotomy was performed on August 29, 1953. Through a combined anterior and posterior approach, numerous loose bodies com-

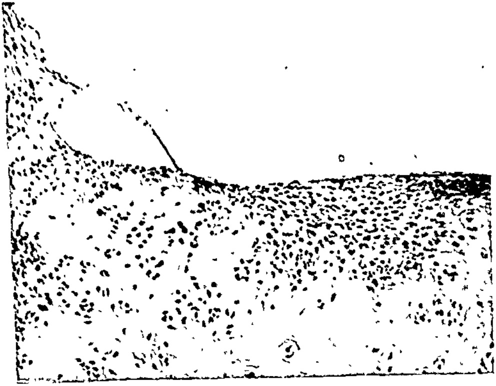


FIG. 4. Second admission. Photomicrograph of section, showing the gradual transition of synovial cells into cartilage cells. The lining cells are intact. × 120.

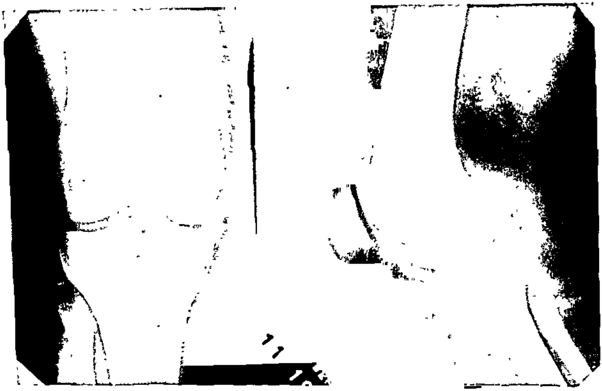


FIG 5. Roentgenogram of the knee, showing a relatively normal appearing joint except for the 2 small round areas of increased density directly anterior to the femoral condyles. There is a suggestion of a soft tissue mass in this same area, but it is nonspecific



FIG 6. Pneumarthrogram of the knee. The quadriceps pouch fills well, but the lower and medial one half of the knee joint fills poorly, and reveals a definite space-occupying intra-articular mass.

posed of hyaline cartilage were removed. A complete synovectomy was done, and the patient made an uneventful recovery. Again the pathologic diagnosis was synovial chrondromatosis (Fig. 4).

When last seen, 18 months after the second operation, the patient exhibited a functional range of motion of the elbow, which occasionally was painful, but only during damp or cold weather. There was no evidence of recurrence.

CASE 2. A 37-year-old white female was admitted to the hospital on December 10, 1951. She had complained of pain and swelling of the left knee of 3 years' duration She had consulted several physicians during that time; all had made a diagnosis of rheumatoid arthritis, for which she had received numerous forms of antirheumatic therapy, none of which helped There was a past history of rheumatic fever with mitral valve involvement Roentgenograms taken elsewhere were reported as negative.

On admission the examination revealed, in addition to a well-compensated mitral stenosis, that the left knee was painful, boggy and swollen Knee motion was limited to a range of 90° (160-70). Routine roentgenograms of the knee (Fig. 5) disclosed a soft tissue enlargement with a small area of calcification in the infrapatellar region. A pneumarthrogram was performed and revealed a soft tissue mass on the medial side of the knee joint (Fig. 6). A diagnosis of synovial tumor was made.

Arthrotomy of the left knee was performed on December 11, 1951. Through a long median parapatellar incision, the synovial membrane was opened, and multiple cartilaginous masses that filled the entire medial aspect of the joint were encountered (Fig. 7). Varying in size from that of a split pea to that of a walnut, they appeared to be attached to the infrapatellar fat pad and to arise directly from the synovial lining. Therefore, a complete synovectomy was done Postoperative convalescence was uneventful The pathologic diagnosis was synovial chondromatosis (Fig. 8).

The small area of increased density seen in the roentgenogram actually suggests a radiologic

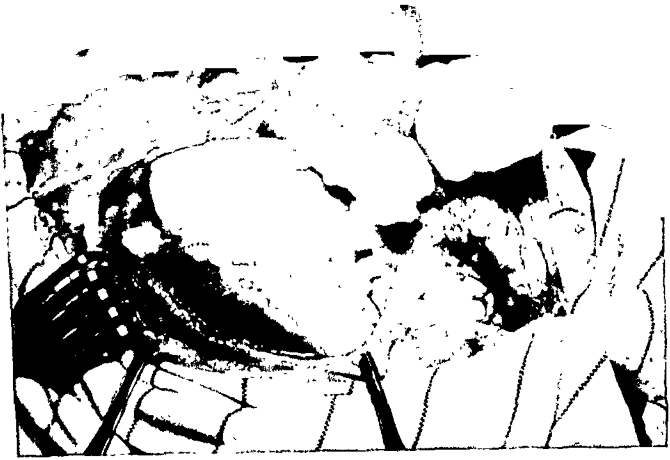


FIG. 7. Photograph at surgery, showing the large masses of cartilage that were encountered on opening the joint.



FIG 8. Photomicrograph of section, which shows a histologic picture similar to that seen in Case 1. A small nest of cartilage can be seen protruding into the synovial cavity; it may eventually have formed a loose body. × 100.

diagnosis of synovial osteochondromatosis; however, histologically this was simple calcification of cartilage, and no true bone was found. Because of the proportionately large masses of uncalcified cartilage present, which were the real cause of the patient's symptoms, it was felt justifiable to label this a case of synovial chondromatosis.

Examination 2½ years after surgery revealed no apparent recurrence. It is an essentially normal knee, with a satisfactory range of motion, and the patient is able to perform her duties as a housewife without difficulty.

DISCUSSION

Although they are varieties of the same disease and produce practically identical symptoms, synovial *chondromatosis* offers a greater problem of diagnosis than does synovial *osteochondromatosis*. In the latter, a diagnosis can be made readily by routine roentgenograms, whereas in the former conventional roentgenograms usually are noncontributory.[11] The 2 cases of synovial chondromatosis which are presented illustrate this point; and they also show that, to the uninitiated, the negative roentgenogram actually may tend toward a misdiagnosis, such as rheumatoid or tuberculous arthritis. It should be emphasized that when confronted with a solitary, chronically enlarged joint of the extremities, one should consider the diagnosis of synovial chondromatosis. Pneumarthrograms can be employed as an aid in arriving at the correct preoperative diagnosis.

SUMMARY

1. Synovial chondromatosis and synovial osteochondromatosis are varieties of the same disease. The cause is unknown. Prevailing theories of etiology are mentioned briefly.

2. The pathologic, the clinical and the roentgen pictures are described. A distinction is made between loose bodies which are calcified or ossified and those which are not. The former condition is called synovial osteochondromatosis, the latter, synovial chondromatosis.

3. Synovial chondromatosis provides a problem in diagnosis because the plain films usually are noncontributory. The pneumarthrogram was used to suggest the preoperative diagnosis of synovial chondromatosis in 2 cases.

REFERENCES

1. Curr, J. F.: Synovial osteochondromatosis and its relationship to the chronic rheumatic diseases of the large joints, Rheumatism 7:209-215, 1951.
2. Freund, E.: Chondromatosis of the joints, Arch. Surg. 34:670-686, 1937.
3. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, ed. 3, p. 699, Philadelphia, Lippincott, 1949.
4. Jones, H. T.: The histogenesis of cartilage as shown in chondromatosis of the knee joint, J. Bone & Joint Surg. 9:310-314, 1927.
5. ———: Loose body formation in synovial osteochondromatosis, with special reference to the etiology and pathology, J. Bone & Joint Surg. 6:407-458, 1924.
6. Kolliker, A.: Human Microscopical Anatomy, translated by G. Busk and T. Huxley, edited by J. DaCosta, pp. 296-297, Philadelphia, Lippincott, Grambo & Co, 1854.
7. Kroh, F.: Die totale und subtotale Synovektomie bei chronischen Synovitiden und generalisierter Chondromatosie, Chirurg 21:1-10, 1950.
8. Mallory, T. B.: Discussion of Case Record #28122, Massachusetts General Hospital, New England J. Med. 226:500-503, 1942.
9. Mussey, R. D., and Henderson, M. S.: Osteochondromatosis, J. Bone & Joint Surg. 31-A:619-627, 1949.
10. Palmer, I.: On the injuries to the ligaments of the knee joint, Acta chir. scandinav., vol. 81, supp. 53, 1938-1939.
11. Timbrell-Fisher, A. G.: A study of loose bodies composed of cartilage or of cartilage and bone occurring in joints. With special reference to their pathology and etiology. Brit. J. Surg. 8:493-523, 1921.
12. Vandemoortele, M. H., Ghormley, R. K., and McDonald, J. R.: Synovial chondromatosis of the ankle joint: report of a case, Proc. Staff Meet. Mayo Clin. 28:437-440, 1953.

Chondromatosis Synovial; Reporto de Duo Casos

Summario in Interlingua

Chondromatosis synovial es un forma de osteochondromatosis synovial. Illo es un condition que affice le membranas synovial de articulationes e bursas. Su occurrentia ha etiam essite reportate, ben que rarmente, in vainas tendinose. Le causa es incognite. Le theorias etiologic que ha essite proponite non esseva acceptate de maniera general. Certe casos es auto-limitante, sed del altere latere il ha reportos de casos in que le disveloppamento de chondrosarcoma esseva observate. Le disordine principal es un proliferation anormal de cartilagine in le revestimento del cavitates synovial. Le chondrogenese de iste cartilagine es un recapitulation del genese observate in le disveloppamento de cartilagine in le embryon. In ver chondromatosis synovial, le processo nunquam continua ultra le stadio del formation de cartilagine. In casos de osteochondromatosis del altere latere le processo pote progreder al formation de osso genuin. In ambe casos le symptomas precipitante le consultation medical es usualmente dolores, tumescentia, e limitate mobilitate del articulation afficite. A vices un historia de blocage articulari pote esser establite, e a vices le prime symptoma notate per le patiente es le presentia de un massa mobile intra le articulation. Le declaration de iste symptomas es usualmente insidiose.

Le aspectos roentgenologic varia ab un caso al altere. Illos depende de si le massas cartilaginose es devenite calcificate o ossificate o si illos ha retenite lor composition purmente cartilaginose. In le prime de iste casos, le roentgenogramma revela le presentia de corpores calcific o ossific intra le articulation. In le secunde caso, le roentgenogramma pote esser completemente normal. Quando isto occurre, pneumarthrographia pote esser de adjuta in corroborar le diagnose de chondromatosis synovial.

Es presentate duo casos, le un con chondromatosis del cubito, le altere con chondromatosis del genu. In ambe casos, pneumarthrographia esseva de valor in establir le correcte diagnose preoperativemente. Le tractamento usate in ambe casos esseva complete synoviectomia.

13

Chondroblastoma of the Talus; a Case Report

LOUIS W. BRECK,* M.D., AND JOHN E. EMMETT,* M.D.

In 1931, Dr. Ernest A. Codman, of Boston, first described this tumor, and it has been called a Codman tumor ever since. He described 9 cases, all of which involved the upper end of the humerus. Because of this, he felt that the location at the upper end of the humerus was a characteristic of the tumor. However, as time has gone by this has not proved to be so; cases have been reported in which various other parts of the body were affected. Even so, the upper end of the humerus remains the most common place of occurrence of this rare tumor. Another characteristic thing about it is that it occurs much more commonly in males than in females. The particular case that we are reporting concerns a female. Typically, these tumors present symptoms first in late adolescence or young adult life. The more modern name for this neoplasm is chondroblastoma, and this term describes the tumor rather accurately.

In 1942, Jaffe and Lichtenstein presented 9 cases of this tumor and wrote an excellent paper on the subject. They clarified the point that this was not a variant of a giant-cell tumor, as Codman had thought originally, but that it originated from growing cartilage cells in the epiphysis The giant cells seen in the tumor had caused confusion, but the cells really were only incidental in these tumors and not the primary cell. They found this to be a disease of adolescence, and they felt that the tumor was benign. The lesion was found to be limited to part of an epiphysis or to involve the epiphysis and part of the adjacent metaphysis, but never to be located in the metaphysis exclusively. Their 9 cases were located as follows: 1 at the head of the humerus, 4 at the lower end of the femur, 3 at the upper end of the tibia and 1 at the lower end of the tibia.

In 1947, Coley and Santoro wrote an excellent article on chondroblastomas and presented a group of 8 cases. One of these cases was located in the talus (astragalus), and, so far as we are able to determine, it is the only one in the literature, besides the case being reported by us, which is located in this bone. Coley and Santoro agreed with Jaffe and Lichtenstein that this tumor was of cartilaginous origin in adolescence, and that it was not related to benign giant-cell tumor of the bone. They (Coley and Santoro) felt that central chondromas were related closely to chondroblastomas. They pointed out several other things. Pain and swelling are the most frequent symptoms of this tumor. Surgical removal of the tumor probably is the best method of treatment in most cases. Tumors of cartilaginous origin usually remain benign, but they may become malignant and metastasize later. Because of this, a chondroma never should be regarded as an inconsequential condition that can be ignored with safety. Vigorous treatment should be instituted, preferably surgical removal. The group of 8 cases reported by Coley and Santoro were located as follows: femur 3, humerus 2, tibia 1, metacarpal 1 and talus 1.

In 1949, Copeland and Geschickter re-

* El Paso, Texas

ported a group of 10 cases of chondroblastoma and commented extensively on this tumor. They were in essential agreement with preceding authors as to the classification of the tumor. They felt that there were benign and malignant variations of this neoplasm. According to these authors, biopsy followed by an accurate diagnosis is very essential. They advised roentgen therapy as the procedure in these tumors before surgical excision. It was their opinion that these neoplasms were chondroblastic tumors that arose from proliferation of cartilage at the epiphysial line at the age of puberty. They pointed out that this tumor was extremely rare, and that the malignant variant of it required very radical resection. The group of 10 cases that they reported were located as follows: lower femur 3, upper humerus 5, lower radius 1 and upper tibia 1.

CASE REPORT

A 21-year-old white female, married, was referred to this office in November, 1952, because of complaints of pain and swelling of the right ankle.

About 2 years prior to her first visit she had sprained her ankle; this failed to improve in the normally expected time. The swelling persisted, and, during the 6 months immediately prior to her first visit to us, the pain had become more noticeable on all motions of the ankle joint.

Physical examination revealed a well-developed and well-nourished person. Pertinent findings were restricted to the right lower extremity. The right ankle was diffusely swollen and tender. Severe pain was experienced on motion in all directions

Roentgenograms revealed a destructive neoplastic lesion involving the talus of the right foot (Fig 1).

The diagnosis at this point rested between a tumor and tuberculosis, and biopsy was advised. This was done, and the tumor was submitted to the pathologist for diagnosis. The first opinion was that the lesion represented a "giant-cell tumor with chondrification." Because of the possibility that this tumor actually was a benign chondroblastoma, as suggested after review of the slides in this office, the slides were sent to Dr. Fred Stewart at Memorial Hospital in New York City. He reported that the tumor was a typical benign chondroblastoma (Fig. 2).



FIG. 1. Preoperative roentgenogram showing anteroposterior view of talus with destructive lesion involving most of the bone.

Approximately 1 month after the biopsy was performed a second operation was done. At this time it was found that almost the entire astragalus was involved by the tumor. In view of this, it was elected to do an astragalectomy, as this offered the only method of removing the tumor completely. This was accomplished without difficulty.

The patient's postoperative convalescence was uneventful, and she was followed thereafter at monthly intervals. The ankle responded surprisingly well, and quickly became relatively painless The patient soon walked with little difficulty. At the end of a year she had continued to do very well without her astragalus, and there was no indication for further operative intervention. Roentgenograms revealed no evidence of recurrence of the tumor.

At the end of 2 years, the patient had continued well clinically, and there had been no change in her ability to walk. She exhibited

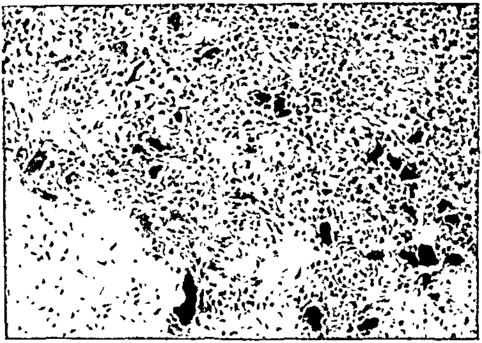


FIG. 2. Photomicrograph of the tumor showing the characteristic appearance of a chondroblastoma. (Dr. Frederick P. Bornstein, Pathologist at Providence Memorial Hospital, El Paso, Texas)

slight calcaneous deformity, but this was not very disabling or unsightly. New roentgenograms revealed no recurrence of the tumor and no arthritis in the ankle.

In March, 1955, the patient was seen again, this making a 2½-year follow-up. She was able to walk long distances and exhibited a barely perceptible limp. She was able to square-dance, and reported that the ankle hurt a little, but not much. Examination at that time revealed minimal swelling of the ankle and good motion. Follow-up roentgenograms once again revealed no evidence of tumor recurrence and only minimal arthritic changes (Fig. 3). A fusion operation was mentioned to the patient, but she would not consider any further operative procedure. She could do all her own housework, square-dance occasionally and walk enough to satisfy her wishes. She was rearing a family of 2 children as a rancher's wife without undue difficulty.



FIG. 3. Roentgenogram showing lateral view of ankle joint 2½ years after astragalectomy for chrondroblastoma of talus.

COMMENT

This case is interesting from several standpoints. The simple removal of the astragalus apparently has effected a cure. The fact that at present the patient is entirely satisfied with the result of her astragalectomy is unusual. It is remarkable in view of the very active life that she is leading. Other unusual features of this case include the sex of the patient, as most of these tumors occur in males, the onset of symptoms following a sprained ankle at a rather later than average age, and the location.

BIBLIOGRAPHY

Codman, E. A.: Epiphyseal chondromatous giant cell tumors of upper end of humerus, Surg., Gynec. & Obst 52:543-548, 1931.

Coley, B. L., and Santoro, A. J.: Chondromas and chondroblastomas of bone. Surgery 22:411-423, 1947.
Copeland, M. M., and Geschickter, C. F.: Chondroblastic tumors of bone; benign and malignant. Am. Surgeon 129:724-735, 1949.
Jaffee, H. L., and Lichtenstein, L.: Benign chondroblastoma of bone, reinterpretation of so-called calcifying or chondromatous giant cell tumor. Am. J. Path. 18:969-991, 1942.

Chondroblastoma del Astragalo; Reporto de Un Caso

Summario in Interlingua

Le tumor cognoscite como chondroblastoma esseva primo describite per Codman in 1931 como lesion del extremitate superior del humero. In le passato illo ha essite confundite con tumor a cellulas gigante, sed le duo es integremente distincte.

Chondroblastomas occurre in sitos altere que le humero. Ben que illos es rar, il ha nunc numerose discriptiones de lor occurrentia.

Condroblastomas es multo plus frequente in masculos que in femininas. Illos occurre durante le adolescentia e ha lor origine in le crescente cartilagine del epiphyse. Il existe un variante maligne de iste neoplasma.

Le tractamento indicate es complete excision chirurgic.

Es reportate un caso de chondroblastoma del astragalo in un femina de 21 annos de etate. Illo esseva tractate satisfacentemente per excision total del astragalo. Le astragalecto mia esseva sequite per inusualmente bon resultatos. Observationes post-operatori durante duo e medie annos ha demonstrate un function satisfactorie e nulle signo de recurrentia del tumor.

14

Bone Changes in the Blood Dyscrasias*

ARTHUR A. THIBODEAU, M.D., AND JOSEPH K. MALOY, M.D.

Changes in the bone structure are of common occurrence in the blood dyscrasias. The skeletal lesions may simulate bone tumors. Correlation of the blood disorders with specific effects on bone may plausibly assist the diagnosis.

In general, the bone marrow bears the brunt of the pathologic changes produced by diseases of the white and the red blood cells. Clinical symptoms and bone changes on roentgen examination are associated invariably with a marrow disturbance. The initial complaints of these patients are usually those of systemic disease such as malaise, weakness, abnormal bleeding and pallor. In some patients, however, it is bone pain, fracture or deformity of the spine or the extremities that occurs at the onset, and only later do the more common symptoms develop; in these, roentgen studies indicate the probable cause of the symptoms and hasten accurate diagnosis by specific studies of the peripheral blood and bone marrow.

RED CELL DISORDERS

The chronic hemolytic anemias, which include Mediterranean anemia, sickle cell anemia and congenital hemolytic jaundice, are characterized by excessive destruction of red blood cells, hyperplasia of the bone marrow and hereditary (racial or familial) transmission of the disorder. Skeletal changes are most frequent and most apparent in the first of these, less so in the second, and uncommon and mild in the third.

In all three of these anemias the basic fault is within the erythrocyte. The abnormal red cell is extrasensitive to trauma of all kinds—mechanical, chemical or changes in osmotic equilibrium. Its lifespan is considerably shorter than the normal of 100 days, regardless of the medium in which it circulates. Normal red cells survive well in the blood *circulation of anemic patients*, but transfusion into the circulation of a normal person does not increase the life duration of these abnormal cells. Their short lifespan strains not only the mechanism for disposal of erythrocytic debris but the mechanism of cell production for replacement. Whether the erythrocyte is defective or deficient is not known precisely. The cause may reside in the hemoglobin molecule. Recent studies show that hemoglobin is of at least 8 different types.[4] It is provocative to speculate that genetically conditioned abnormalities of its synthesis may determine the kind of hereditary hemolytic disease that develops.

MEDITERRANEAN ANEMIA

Mediterranean anemia is known also as Cooley's anemia, familial erythroblastic anemia, and thalassemia. The majority of cases occur in people of Mediterranean stock, but a similar anemia has been reported in natives *of China and Thailand.*[5] In this blood dyscrasia the erythrocyte is deficient in the nor-

* From The New England Center Hospital, Boston, Mass The authors are indebted to Dr. William Dameshek, Chief of the Hematology Service, for use of his case material.

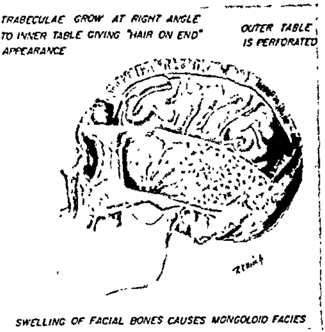


FIG. 1. Mediterranean anemia. Enlargement of the facial bones is particularly marked.

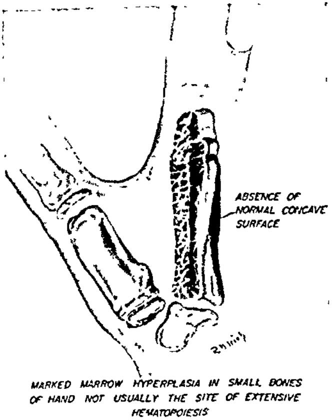


FIG. 2. Mediterranean anemia. There are coarse trabeculation and loss of normal concave surfaces of the metacarpals.





mal type of blood pigment because of the impaired affinity of the hemoglobin molecule for its essential constituent iron. The peripheral blood reveals a hypochromic microcytic anemia and a high serum iron. There are wide variations in the size and the shape of the red cells, and flattened *target* cells are seen in the stained smear. These *thin* cells can withstand solutions of hypotonic saline that would cause swelling and rupture of the

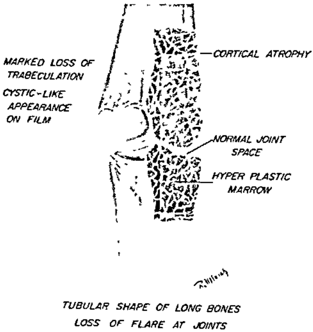


FIG. 3. Mediterranean anemia.



normal red cell, which accounts for the diminished osmotic fragility that is a feature of the disease. Sickling does not occur. The clinical range covers the spread from a severe anemic state to a minor hematologic abnormality.

Bone Changes. In severe cases, extreme marrow hyperplasia occurs, and all bones become the site of erythropoiesis. The bone marrow exerts constant pressure in all directions, and erosion and deformation of both spongy and cortical bone develop. In the skull the tables are widely separated, the outer table badly atrophied and scarcely visible by roentgenogram. In cases of severe involvement, the development of the trabeculae of the diploe at right angles to the tables gives rise to a striking hair-on-end appearance. Enlargement of the zygomas causes prominence of the cheek bones and facies of a mongoloid type. This is complicated further by distortion of the maxillae, with resulting malposition of the teeth and malocclusion of the jaws.

The changes in the long bones are dramatic. The marrow cavity widens, the cortex thins, the mid-shaft loses its concave borders, and the ends of the bone become rectangular (Erlenmeyer flask deformity) The substantia spongiosa usually is coarsely trabeculated, and near the ends of the diaphyses there are dense horizontal striations suggestive of growth lines During childhood these changes are progressive. If the patient survives to young adulthood, some areas sclerose. The sclerotic bone may ring areas of translucence, and the lesion then resembles the bone infarcts of caisson disease. In the severe case, both maturation and over-all growth of the skeleton are retarded, and the victim is definitely smaller than normal.

Treatment. Splenectomy, iron therapy and the newer vitamin and steroid preparations are of no benefit. Blood transfusions ameliorate the acute attacks.

SICKLE CELL ANEMIA

Sickle cell anemia seems to be peculiar to Negroes and only to those whose erythrocytes exhibit the sickling trait. The sickling abnormality is present in about 7 per cent

of the Negro population of this country, but a frank case of the disease occurs in fewer than 0.2 per cent. Reduction of the patient's oxygen tenison to levels of 35 or 40 mm. of mercury brings out the characteristic sickle shape in the red blood cells. In fresh blood these cells revert to normal shape when the oxygen tension is elevated sufficiently. In the absence of the disease, trait-defective cells are less susceptible to anoxia, but they also sickle if the oxygen tension is low enough. Negroes who have sickle cell anemia have recurrent episodes of severe anemia, pain in the limbs and the abdomen, and leg ulcers. Those who have only the sickling trait are healthy, have a normal life expectancy and have no hematologic abnormality on ordinary examination.

Bone Changes. Sickle cell anemia manifests itself at a later age than Mediterranean anemia does. This explains why there are few changes in the bone structure in infants and in children susceptible to the disease; in the long bones, which are especially free of change, it is not until adolescence or adulthood that lesions resembling bone infarcts occur. Infarction develops because low oxygen tension causes a tremendous increase in the viscosity of the blood, stasis of the erythrocytes ensues, and masses of abnormal cells impact the lumens of the smaller vessels of all organs. Postmortem studies show extensive thromboses and numerous areas of infarction resulting from plugged vessels. When this process occurs in bone, the necrotic areas can support no weight-bearing; depending on the site of infarction, kyphosis and scoliosis, or aseptic necrosis of the hip joint, develop. The long bones exhibit thick cortices and narrow medullary canals—just the reverse of that seen in Mediterranean anemia. In the skull, however, the changes in both diseases are similar—widening of the diploic space, atrophy of the tables and thickening of the calvarium.

Treatment. The bone changes demonstrated by roentgen examination are not reversible, since no satisfactory method of treating the blood dyscrasia is known. During the acute attacks, transfusions of whole blood are helpful.

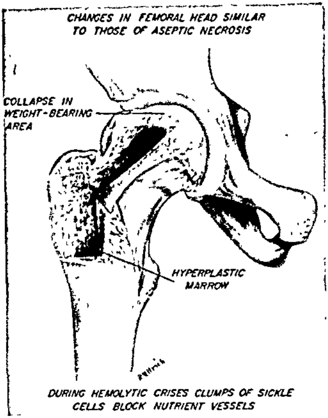


FIG. 4. Sickle cell anemia. This is a complication of bone infarction.



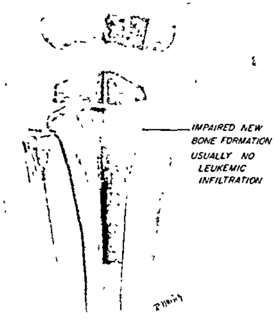


FIG. 5. Leukemia. This demonstrates the transverse band of diminished density at the metaphysis.



CONGENITAL HEMOLYTIC JAUNDICE

Characterized by a broad range of clinical severity ranging from severe anemia with jaundice to the scarcely perceptible case, congenital hemolytic jaundice is known also as familial hemolytic icterus and hereditary spherocytosis. The red cells are *fat* by comparison with the normal red cell, and especially so by comparison with the *thin* red cell of Mediterranean anemia. They are also rounder, and have increased osmotic and mechanical fragility. Filtered out in the spleen, they apparently undergo destruction at that site.

Bone Changes. Bone changes are limited to the skull. They are similar in type to those described for the other hemolytic anemias, but are of lesser degree. The long bones seldom are affected.

Treatment. Splenectomy is of some benefit; after this procedure the osmotic and mechanical fragility of the erythrocytes in the circulating blood declines toward normal.

WHITE CELL DISORDERS

LEUKEMIA

Leukemia is a malignant, invariably fatal, disorder of the blood-forming organs that is characterized by abnormal proliferation of the leukocytes and their precursors. Differentiation of its protean manifestations is based on aggressiveness of the disease and the predominating type of abnormal cell

The acute form of leukemia is associated with a predominance of immature cells. It appears generally in children, and runs a fatal course in less than 6 months The chronic form, in which mature cells predominate, develops more often in adults, and runs a course of from 1 to 3 or 5, and often 10, years. The *subacute* is closer to the acute form, but it may affect either children or adults, and terminates fatally after a course of between 6 and 12 months.

Classification according to cell type involves the stem cell, granulocyte, lymphocyte, monocyte, plasmocyte and thrombocyte. Further complications are presented

when the predominance shifts from one type of cell to another during the course of the dyscrasia; for example, from stem cell to lymphocyte or from granulocyte to monocyte.

The effects of leukemia are both widespread and local.[3] Diffuse involvement of the bone marrow causes a disturbance in both red cell and platelet production. During the early stage of myelogenous leukemia, there may be polycythemia, but anemia develops ultimately in this type, as it does initially in the other types of the disease. Thrombocytopenia is responsible for the hemorrhagic tendency present in many of these patients, especially when the anemia is well defined. Local enlargement of the lymph nodes may cause venous obstruction and edema of the extremities, tracheal stenosis and asphyxia, or it may cause obstructive jaundice by compressing the bile duct system. Leukemic infiltration may occur anywhere in the reticuloendothelial system; impairment of function has been noted in such unrelated areas as the skin, the gums and the peripheral nerves. At postmortem examination the bone marrow, the spleen and the lymph nodes are seen invariably to be grossly or microscopically involved. The normal marrow space is usurped by proliferative masses of abnormal white cell tissue.

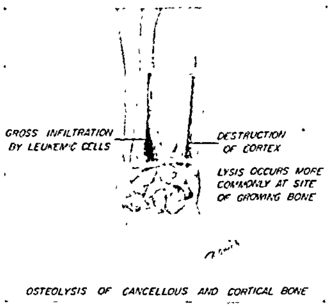


FIG. 6. Leukemia. Osteolysis of the distal ends of the radius and the ulna.



Bone Changes. The possibility of detecting skeletal involvement ante mortem depends on the severity of the disease and the vigilance of the search. Bone changes occur in about 50 per cent of leukemic children, only half of whom complain of bone pain. It is evident that the bone lesion may be silent symptomatically. Acute leukemia in children invariably affects the skeleton unless death occurs soon after onset. This is due to both the aggressive character of the tumor cells and the presence of active marrow in the peripheral as well as the axial skeletal system

The following are the 4 different kinds of bone change whose presence should alert the physician to suspect leukemia as the responsible agent.[6,8] Although none of these changes is pathognomonic of leukemia, any

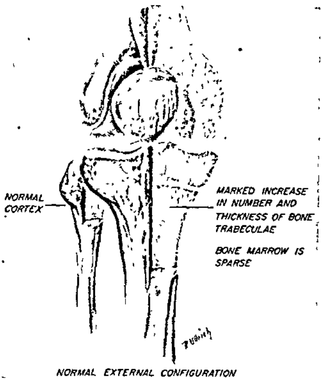
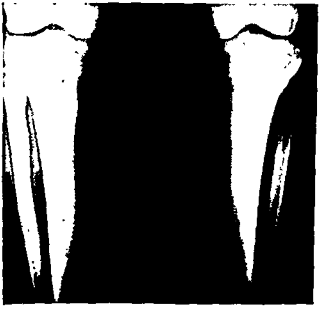


FIG. 7. Leukemia. Osteosclerosis of the femur and the tibia.



one of them is a signal for further study by blood and bone marrow smears.

1. *A transverse band of diminished density in the diaphyses of the long bones* is the least specific of the bone changes. The band is seen most easily in the knees, where bone growth is most rapid. It has increased radiolucency; it may have either sharp or ill-defined borders. Only occasionally does the band itself contain leukemic cells. The defect apparently is a nonspecific response of growing bone to acute systemic illness. A similar picture is seen in acute infections and in malnutrition, as well as in some normal children. The band may disappear completely during a remission, leaving only a horizontal band of dense bone.

2. *Osteolysis* is due to leukemic infiltration of both spongy and cortical bone. The process may be diffuse or focal, and may coexist with the transverse band just described. Some observers have pointed out that leukemia is not as aggressive as the primary or the secondary tumors that affect bone; infarction, necrosis and hemorrhage thin and deform the adjacent bone, but active invasion of bone by columns of malignant tissue, which is seen in other types of cancer, is minimal. The bone, nevertheless, is weakened, although not so soon. Bone tenderness is common, and spontaneous fracture occurs in the long bones and the vertebrae. Roentgenographically, in children these lytic lesions are seen most frequently in the metaphyses of the femur and the tibia, the proximal portion of the humerus and the distal radius. In adults, the lytic changes of chronic leukemia are seen most frequently in the pelvis, the skull and the vertebral column. Sometimes the entire skeleton becomes diffusely osteoporotic.

3. *Osteosclerosis* occurs in about 10 per cent of the patients who have acute leukemia. The involved bones show an increased diffuse radiodensity to roentgenograms. This is attributed to stimulation of the spongy bone by leukemia, which causes an increase in both size and number of bone trabeculae. The bone also is dense on postmortem examination. Since in the early stages the process may not be uniform, patchy areas of sclerosis are interspersed among more normal bone.

4. *Periosteal elevation* results when leukemic infiltrates penetrate the cortex of the long bones. The elevated periosteum reacts by producing layers of new bone beyond the

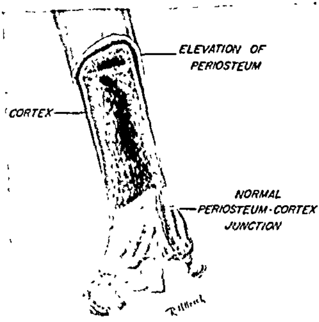


FIG. 8. Leukemia. Elevation of the periosteum of the humerus.

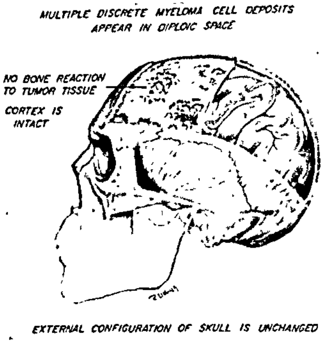
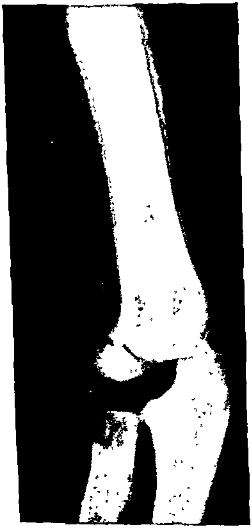


FIG. 9. Multiple myeloma. The skull contains multiple nonreactive areas of bone destruction.





cortical margin. This is typical of about 20 per cent of the cases of acute leukemia.

During remissions of leukemias, either spontaneous or induced, bone lesions of the first 2 types discussed may regress to the point at which the bone appears to be normal on roentgen examination. This effect has

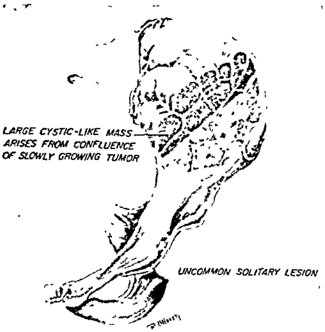


FIG. 10. Multiple myeloma. This is a solitary lesion of the wing of the ilium. *Biopsy was necessary to establish the diagnosis.*



been reported to follow such treatment as local irradiation and the administration of radioactive phosphorous and folic acid antagonists. *No cures have been reported; the* bone lesions recur, although not always in the same area.

Multiple Myeloma

Multiple myeloma often causes bone pain and sometimes pathologic fractures. Most commonly encountered in men over 50 years of age, the disease is associated with anemia, hyperglobulinemia and proteinuria. The derangement of protein levels in the blood and the urine has been so severe in some cases that the disease has been thought to be due *primarily to a fault in protein metabolism.*[7] The protein in the urine may be of the usual type, which is precipitated by ordinary boiling, but about half the patients excrete the special Bence Jones protein, which has to be discovered by careful heating and cooling of the voided specimen, since it precipitates at from 50° to 60° C. and goes back into solution at 100° C. In about half the patients, hypercalcemia is present, associated with metastatic calcification in the soft tissues. In this disease, the peripheral red cells show a degree of anemia in proportion to the amount of marrow space usurped by the myelomatous tissue; the peripheral white cells are within normal limits until the terminal stage.

Bone Changes. Examination of bone marrow aspirations is needed for confirmation of the diagnosis, but if the roentgen findings are at all typical *of the following,* they are highly suggestive of multiple myeloma.

The classic picture is the *punched-out* lesion. There is a well-defined area of radiolucency with a sharp border, but no bone reaction in the surrounding area. The lesion appears to be inert, shows no destructive or deforming properties, is relatively small and characteristically multiple. It is observed best *in the skull, the spine and the pelvis,* where the adult marrow is most active. Contrasted

with this almost pathognomonic picture is the one in which there are only a few vague ill-defined areas of radiolucency or diffuse osteoporosis of the entire skeleton. However, when associated with bone pain and anemia, these findings raise the index of suspicion and make bone marrow studies mandatory. There are pathologic fractures in the long bones and collapse of the vertebral body in areas in which the cortical bone has been weakened severely. When the spinal column is involved, signs of cord damage may reach the stage of paraplegia.

Treatment. Impending or actual fracture of the long bones is treated best by intramedullary fixation.[9] The presence of the metal does not preclude the use of local radiation or any of the therapeutic drugs. The fractures often heal despite the presence of tumor tissue.

Treatment with urethane and stilbamidine has produced clinical remissions but no real improvement in the bone lesions. As is the case with leukemia, no permanent cures have resulted from any kind of therapy. Some of the longest periods of survival have been those of patients who had the solitary lesion of multiple myeloma. There are well-documented cases in which apparently only one area of the body has undergone myelomatous degeneration. Surgical excision of the lesion has produced amelioration for intervals lasting from 10 to 15 years before the more characteristic picture of the disorder has appeared. Roentgenographically, the solitary myeloma may be confused with a benign bone tumor, since destructive or invasive features are absent. Biopsy is necessary to establish the diagnosis.

HEMORRHAGIC DISORDERS

Abnormal bleeding may be due to a defect in the blood vessels, a deficiency of platelets or abnormalities of the coagulation mechanism. Scurvy is an example of the first, and hemophilia is the classic example of the last. Bone changes that are characteristic occur in both of them.

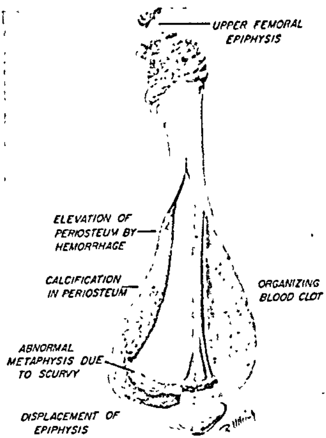


FIG. 11. Scurvy. Healing phase following extensive subperiosteal hemorrhage of the femur.



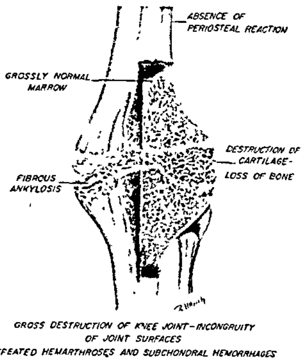
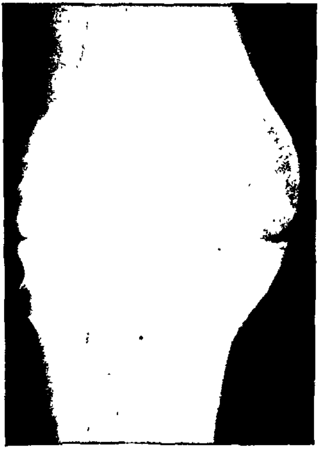


FIG. 12. Hemophilia. Repeated hemarthroses over a 20-year period have resulted in complete disorganization of the knee joint



SCURVY

Vitamin C is essential to the deposition of intercellular collagen in the connective tissues that constitute the walls of the blood vessels. When there is complete absence of this vitamin, the capillaries cannot develop normally. The tourniquet test, which increases the hydrostatic pressure within the capillaries, is positive. The bleeding time, an index of the quantity of blood platelets, is normal; so also is the clotting time, which is indicative of the efficiency of the coagulation mechanism.

Bone Changes. Bone changes in this disease are of 2 basic types:[1] one is caused by defective intercellular substance at the ends of the bones, where growth takes place; this results in the development of multiple spurs at the cartilage-shaft junction, subepiphysial atrophy and fractures through the epiphysial plate. The other is caused by abnormal bleeding. Subperiosteal hemorrhage is common. Seen most frequently in the long bones, such as the femur, the tibia and the humerus, it sometimes involves the entire length of the bone. During the acute bleeding phase, there is extensive swelling of the soft tissue, owing to the large hematoma and to edema of the overlying tissue. During the healing phase, a smooth shell of bone appears along the periphery of the hematoma, well outlined against a background of soft tissue. As the hematoma is resorbed, this layer of bone recedes to meet the shaft of the long bone, where it becomes the new cortex. Hemorrhage into a joint is exceedingly rare in infants and children, but why is not known.

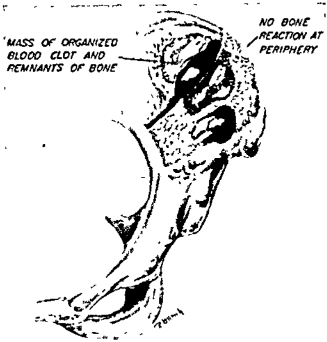
HEMOPHILIA

The hemorrhagic diathesis is transmitted through the mother and inherited by only the male progeny Hemorrhage occurs in any part of the body; it arises either spontaneously or following trivial trauma. The clotting time in these patients is distinctly prolonged, seldom being less than 30 minutes. The blood vessels are normal, and there is

no diminution in the number of platelets. It is unlike scurvy in that hemarthrosis is common and subperiosteal hemorrhage is rare. Recurrent episodes of bleeding into the joints lead to chronic deformity and disability. Minor cuts and tooth extractions are catastrophic events in the life of a hemophiliac; contusions cause extensive bleeding into subcutaneous tissues and muscle. These are serious problems when they occur in areas in which the mass of blood embarrasses respiration and circulation. During acute bleeding, immobilization of the area involved and transfusions of fresh blood or plasma will speed cessation of further hemorrhage.

Bone Changes. In hemophilia, some bone changes result from the extravasation of blood into the bone, and others are progressive changes secondary to repeated hemarthroses.[2] Bleeding into the bone at the joint margin causes irregular subchondral defects. This lesion is not nearly as serious as the more common hemorrhage into the joints. Blood within a joint is absorbed after a variable length of time, depending on the extent and the condition of the synovial lining, and recovery of joint function and appearance is complete in most instances. It is only when there are recurrent episodes of bleeding that the blood is absorbed incompletely. The residual blood then sets up a chronic inflammatory reaction that leads to loss of motion, permanent deformity and fibrous ankylosis Atrophy of muscle and bone is concomitant with the loss of joint motion

During acute hemarthrosis, the joint is hot, red, painful and swollen. These signs subside slowly, and joint function improves; after many recurrences of hemorrhage over the years, enlargement of the joint becomes permanent. Synovial hyperplasia is prodigious Synovial encroachment destroys the articular cartilage, the joint space narrows, and its central portion is invaded by connective tissue that does not stop at the bone edge but goes on to invade the subchondral layer. Radiologically, there is much narrow-



HEMOPHILIA SIMULATING TUMOR OF ILIUM

FIG. 13. Hemophilia. Uncommon lesio simulating a bone tumor.



ing, or even obliteration, of the joint space; irregular marginal defects exist at the site of the former joint line; the periarticular soft tissue is swollen; and the surrounding bone is atrophied diffusely. The outlook for joint function is poor when this stage has been reached.

CONCLUSION

The bone changes that occur during the course of the blood dyscrasias vary considerably as to site, frequency and distribution. The chronic hemolytic anemias are characterized by the diffuse nature of the alterations in the bone. In the white cell and hemorrhagic disorders, however, the lesions may be of the diffuse or the focal type, depending upon the stage and the severity of the disease. It is in the latter kind that confusion with a bone tumor, either benign or malignant, may arise, and awareness of this fact assists in making the correct diagnosis.

REFERENCES

1. Caffey, J. P.: Pediatric X-ray Diagnosis, p. 704, Chicago, Yr. Bk. Pub., 1945.
2. *Ibid.*, p. 736.
3. Castle, W. B.: Disorders of the Blood *in* Pathologic Physiology; Mechanisms of Disease, Philadelphia, Saunders, 1950.
4. Chernoff, A. I.: The human hemoglobins in health and disease, New England J. Med. 253:322, 1955.
5. Silver, H. K: Mediterranean anemia in children of non-Mediterranean ancestry, A.M.A. Am. J. Dis. Child. 80:767, 1950.
6. Silverman, F. N.: Skeletal lesions in leukemia, Am. J. Roentgenol. 58:819, 1949.
7. Snapper, Isidore Metabolic Bone Diseases; Facts and Theories *in* The Musculoskeletal System, New York, Macmillan, 1952.
8. Sussman, M L · Skeletal changes associated with diseases of the blood, Bull New York Acad. Med 26:763, 1950
9. Thibodeau, A A., and Maloy, J K.: Management of metastatic bone disease, Bull. New England Med Center 16:108, 1954.

Alterationes Ossee in Dyscrasias del Sanguine

Summario in Interlingua

Alterationes ossee occurre communmente in le curso del dyscrasias sanguinee. Disordines erythrocytic que resulta in anormalitates ossee include anemia mediterranee, anemia a cellulas falciforme, e congenite ictero hemolytic. In iste gruppo de morbos le medulla ossee suffre extreme hyperplasia, con le resultato de atrophia del osso tanto cortical como etiam cancellose. Si le juvene patiente supervive usque al fin del adolescentia, le externe configuration de omne su ossos es marcatemente alterate.

Leucemia produce quatro different typos de alteration ossee. Illos es: (1) Un banda transversal de diminuite densitate al metaphyse, (2) osteolyse, usualmente in le portion crescente del osso, (3) osteosclerosis, e (4) elevation del periosteo. Le presentia de un de iste quatro alterationes indica le necessitate de un studio del sanguine peripheric e del medulla ossee. Myeloma multiple es characterisate per un parve, non-reactive, foraminiforme lesion que se trova le plus communmente in le cranio, le columna vertebral, e le pelve.

Hemorrhagia subperiosteal occurre in scorbuto. Radiographicamente on nota intumescentia del texito molle e le apparition de un strato de osso distantissime ab le cortice durante le phase recuperative. Hemophilia se associa cognoscitemente con hemarthroses de frequente occurrentia, sed hemorrhagia subperiosteal es rar in iste condition. On incontra destruction del superficies articular, erosion de osso subchondral, e diffuse atrophia ossee.

In le disordines del cellulas blanc e del mechanismo de sanguination, il occurre isolate lesiones que resimila tumores ossee.

15

Perineurial Cysts

Review of Literature and Report of Eight Cases

KENNETH H. ABBOTT, M.D.*

Although intraspinal meningeal cysts were described as early as 1898 by Schlesinger,[8] it was not until 1938 that perineurial cysts were described. These were found in cadavers by Tarlov,[13] and 10 years later he described these cysts in life as a clinical syndrome simulating lumbar intervertebral disk protrusions. Further confirmation of this came in reports by Rexed (1947),[7] Weiford (1950)[18] and Schreiber and Haddad (1951).[9] In 1952[16] and 1953,[15,17] Tarlov presented further evidence that perineurial cysts, usually situated in the sacral canal, could produce backache, sciatica, urinary and sexual disturbances, and reflex and sensory changes. These conclusions have been substantiated by Schreiber and Haddad,[9] Weiford,[18] Taheri *et al.*,[11] Strully and Heiser,[10] Jacobs *et al.*,[5] Mérei,[6] and by 3 cases (and probably a 4th case) of the author's and 1 of Dr. Harry LeFever's to be reported here.

At first it was presumed that these lesions could not be diagnosed roentgenographically; however, it has been shown that a spinal subarachnoid contrast media (pantopaque) may enter one or more of these cysts (Taheri *et al.*,[11] Tarlov,[15] Strully and Heiser,[10] Jacobs *et al.*,[5] and Mérei[6]) and thus allow a roentgenographic diagnosis without the necessity of "exploratory" surgery. Furthermore, Browder,[1] Jacobs *et al.*,[5] and Mérei,[6] as well as this study, have shown that erosion of the sacrum or enlargement of the sacral canal (*hollowed-out area*—Browder) with marked thinning of the posterior arch may be seen roentgenographically on the routine films of the sacrum in an occasional case.

Tarlov[12,15,16] believes that these cysts are of traumatic etiology, with a splitting of the nerve-root coverings so that the potential space between the endoneurium (derived from the pia mater) and the perineurium (derived from the arachnoid) is distended to form a true cyst. Serial sections have shown the cysts to begin in the perineurial space, and they may be surrounded by the entire nerve root; or they may invade the nerve root or the posterior root ganglia, and thus the cysts become surrounded by compressed nerve fibers. These nerve fibers, as well as the dorsal root ganglia, have shown extensive degenerative changes. Tarlov[12] has emphasized that:

> trauma, direct or indirect, or any one of the manifest causes of tissue hemorrhage in general may produce bleeding and (results in) permanent residual cyst formation.

It was shown from serial section that hemorrhage spread to a limited extent along the perineurial space and then distended and ruptured veins within the nerve root and ganglion or infiltrated this structure directly. Absorption of the hemorrhage and destroyed nerve tissue leads to cyst formation. The presence of rarefied neural tissue and cavita-

* From the Department of Surgery, the Division of Neurosurgery, College of Medicine, Ohio State University Hospitals, Columbus, Ohio.

Case 1. A 49-year-old male had experienced some low backache off and on for 1½ years after *wrenching* his back when he fell through a trap door and landed on his right hip. Eight years previous to this he had strained his back while lifting a 300-pound pan of metal plates—a short man on the other side dropped his side of the pan. Six months before hospitalization, typical left lower backache began, accompanied by left sciatic pain that spiraled down to the medial aspect of the knee. Dental extractions and osteopathy were of no help. Two months previous to hospitalization, bilateral hip and sciatic pain was more severe on sitting, on arising in the morning (it awakened him in the early morning), on coughing, on sneezing and on straining. He obtained some relief from the use of a hard bed and from walking about (day or night).

Examination disclosed marked splinting of the back muscles with severe limitation of motion of the spine and with a list to the left. Both knee and ankle jerks were absent. Hyperalgesia was present over the right S-1 dermatome, and slight hypalgesia was noted in areas over the left S-1-2-3 dermatomes The spinal fluid contained 30 mg. per cent protein, and the rest of the examination was normal. The myelogram (Fig. 1 A-D) disclosed bilateral sacculation of the pantopaque in the S-1 and 2 roots consistent with that seen in perineurial sacral cysts. Typical thin-walled translucent cysts with nerve fibers in their walls were found on S-1 and S-2 roots at surgery (July 12, 1949), as well as smaller cysts on the S-3 roots. These were treated by incision and then imbrication with No. 5-0 silk sutures and covering with Gelfoam. Inasmuch as there were degenerative changes of the lumbosacral disk, an *onlay* Hibbs type of fusion was done (by Dr. Henry Lacey).

The patient has made a complete recovery. He is able to do heavy work and rarely has any discomfort. He was last seen in March, 1955.

Case 2. A 53-year-old female was seen March 5, 1954, because of "incontinence of urine, weakness of the lower extremities (the left worse than the right), tingling paresthesia in the left lower extremity and a dull ache in the lower back." All symptoms began following an injury to her back on January 8, 1953. There had been only mild progression of her symptoms after they became well established. The neurologic examination revealed a left Horner syn-

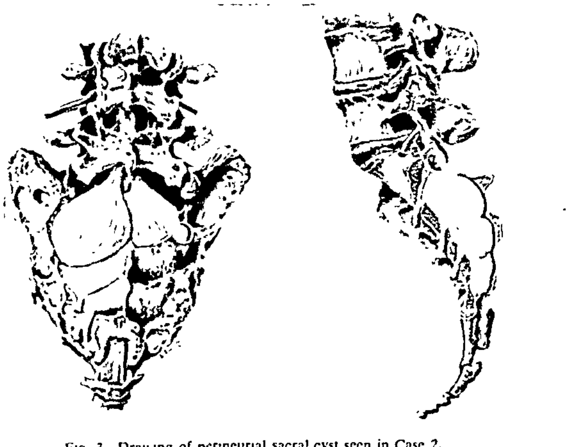


FIG 3. Drawing of perineurial sacral cyst seen in Case 2.

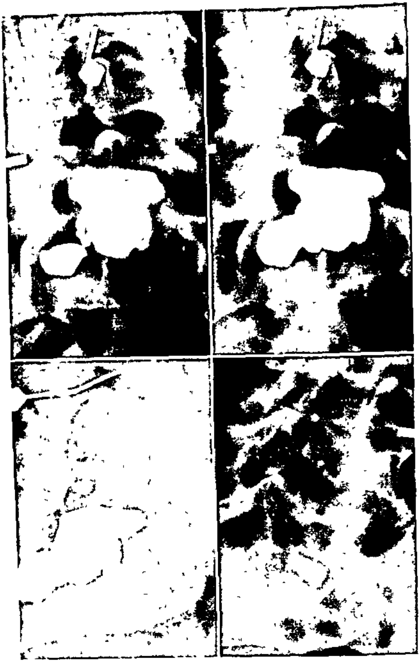


FIG. 4. *(Top, left & right, and bottom, left)* Multiple views of pantopaque myelogram in the lumbosacral area disclosing 1 perineurial cyst on the right S-1 root. A droplet of "dye" appears loculated at L 4-5 interspace, suggesting a possible tiny arachnoidal pouch at this level. *(Bottom, right)* Oblique view taken after removal of the dye with residual dye in the S-1 cyst.

drome, markedly reduced ankle jerks and an unsteady gait apparently due to pelvic girdle weakness.

urina[illegible]

sion over the upper sacral and the lower lumbar spines elicited distress. Plain films of the sacrum disclosed a markedly enlarged sacral canal. A pantopaque myelogram (Figs. 2 & 3) disclosed rather large arachnoidal cystic defects on S-1 and S-2 roots and to a lesser degree on others. At surgery, April 15, 1954, the left side of the paper-thin posterior arch of the sacrum was opened, revealing large cysts on S-1, S-2 and S-3 roots with nerve filaments coursing through the walls of these cysts. With only minor technical difficulties caused by epidural venous hemorrhage, the cysts were handled easily by means of imbrication with No. 5-0 silk sutures and by covering them with Gelfoam.

The postoperative course was not unusual, but the patient has been left with considerable sensory deficit in S-2 and S-3 dermatomes and with increased sphincter weakness.

Case 3. A 76-year-old white male was admitted to the hospital July 7, 1954, because of 5 weeks of right sciatic pain. Some months before this he may have wrenched his back. Soon after the pain commenced he noticed a right footdrop. The ipsilateral knee and ankle jerks were absent. A myelogram (Fig. 4) revealed a small perineurial sacral cyst at the S-1 level on the right side. At surgery a small perineurial cyst about 1 cm. in diameter was found and removed

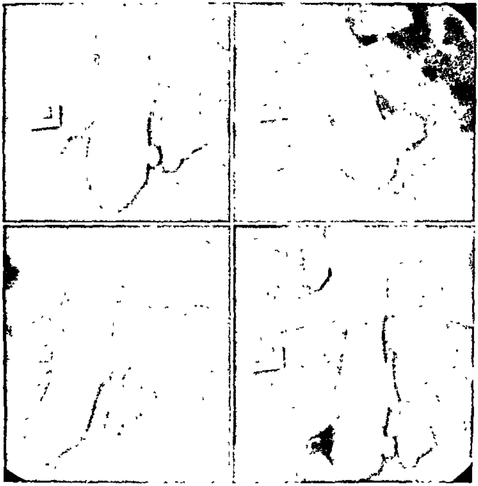


Fig. 5. Myelogram with pantopaque filling solitary (perineurial?) cyst.



Fig 6 *(Left)* Anteroposterior view of sacrum disclosing the spina bifida occulta 1-5 and the markedly enlarged sacral canal with apparent erosion (?) in the right half of the canal *(Center & right)* Anteroposterior and oblique views with pantopaque present in the large sacral canal cyst. (Perineurial cyst or sacral meningocele?)

carefully but subtotally. The rest of the wall was imbricated with No. 5-0 silk. The posterior root ganglia of S-2 and 3 were found to be larger than normal and appeared to be inflamed.

Case 4. A 67-year-old white male, a carpenter, was seen March 11, 1955, because of left sciatic pain of 6 weeks' duration. In November, 1954, while lifting a long board, one end of which was on a trestle, the board slipped and his lower back suffered a snapping, flexion injury. The pain appeared immediately after this in the posterior aspect of the left lower extremity and was accompanied by numbness of the sole of the left foot. Cough, sneeze and strain aggravated the discomfort.

A detailed neurologic study failed to disclose any abnormalities except for a positive Lasègue sign at 45° on the left.

Roentgenographic lumbosacral films were not unusual for one of the patient's age. A pantopaque myelogram (Fig. 5) disclosed a sacral cyst at S-1 on the right.

Following myelography, the patient stated he felt *cured* and wanted to be released. Four weeks later he was still asymptomatic and anxious to return to work, hence surgical confirmation of this roentgenographic evidence has not been possible.

Case 5. On June 20, 1954, a 28-year-old white female was admitted to the services of Dr. H. E. LeFever at the University Hospital. She had experienced urinary and rectal incontinence since childhood and weakness of her lower extremities since she was 16 years of age. On examination it was found that the deep tendon reflexes in the lower extremities were absent, which was associated with hypasthesia and hypalgesia in the sacral dermatomes—more severe on the left side. A high steppage gait prevented her from climbing stairs without the aid of her hands to pull her up by a handrail. There was considerable muscle atrophy in the lower extremities and the buttocks.

Roentgenograms of the sacrum revealed a very large sacral canal with obvious thinning of the posterior bony plate. A myelogram (Fig. 6) disclosed evidence of a large cyst that occupied the sacral canal There was a small opening between the cyst and the cul-de-sac of the normal lumbosacral leptomeninges

The operation performed by Dr. LeFever is reported as follows:

"The spine and the lamina of 5 were removed, as well as the thin layer of bone overlying the sacral hiatus. It was immediately apparent that a large sacral cyst was present, arising from the inferior portion of the cul-de-sac. The whole cyst was resected free, and this occupied the left side of the dural sac and extended inferior to the cul-de-sac. The opening was at the extreme end of the cul-de-sac. The whole cyst was removed, and the dura was opened in order to be certain that this represented the only opening. The filum terminale was sectioned, and the dura was repaired. The cyst extended from the 1st to the 4th sacral segments, greatly enlarging the sacral canal."



FIG. 7. Photomicrograph of cyst removed in Case 5. The cyst wall appears to be made up of collagenous and fibrous tissue with some areas of "pavement" type cells on its inner surface.

The patient now walks with a waddling gait, but she is working and has no sphincter incontinence and no pain or distress.

COMMENT

It is to be noted that all 5 cases were diagnosed before operation as having arach-

noidal cysts, but only 4 have been proved surgically. In retrospect, at least 3 of these (Cases 1, 2 and 3) can be diagnosed roentgenographically as presenting perineurial sacral cysts. The diagnosis in Case 5 still is not clear; some may question this diagnosis, inclining toward its being a congenital intraspinal meningocele. The histologic structure of the cyst shown in Figure 7 is inconclusive. The presence of collagenous and fibrous tissue, *pavement-type* cells and possible nerve fibers suggests it to be a perineurial cyst. However, it is recognized that none of these findings is definite enough to prove its identity beyond question.

Two of these 5 cases gave a distinct history of trauma to the lower back. In Case 3 the story of injury was not clear. In this case the noncystic dorsal root ganglia seen at operation appeared to be swollen, as if caused by an inflammatory process, though trauma (hemorrhage) could not be ruled out. In Case 3 the absence of patellar tendon and tendo achillis reflexes on the right and bilateral absence of both in Case 1 were confusing in the presence of a sacral canal lesion. It may be that this indicates a more widespread involvement of nerve roots (including L-3 and/or L-2?) than the sensory, the motor or the myelographic examination of these patients would suggest. The small droplet of opaque media seen in the two anteroposterior views (Fig. 4 A-D) at the upper lateral border of L-5 suggests the presence of some dilatation of the L-5 nerve root *sleeve*, which could be the beginning of a perineurial cyst (?). We have added here 2 cases with multiple cysts to the 4 that Tarlov has reported. It seems surprising that there are not more with multiple cysts.

DISCUSSION

Perineurial cysts undoubtedly occur more frequently than the few reported cases would indicate. The author has encountered myelographic evidence of 3 cases (Case 4 and two others) with small intrasacral cysts associated with low back and sciatic pain. The symptoms have been intermittent and, so far, have not come to surgical confirmation.

The sacral canal still remains a relatively unknown area, and it would appear that it may well be of importance in the cause of *disk-like symptoms*, second only to the position now held by the lumbar disks themselves. When symptoms point to sacral root disease, more frequent attention must be given to the minor or less evident sacral cul-de-sac *pantopaque* anomalies. This may well lead to many negative explorations of the sacral canal. On the positive side, however, it may lead to finding the cause of *disk symptoms* when there is no roentgenographic or surgical evidence of a disk protrusion (Schreiber and Haddad,[9] Tarlov,[15] Strully and Heiser[10]). A thorough search under the sacral arch when a negative disk exploration is encountered may prove to be fruitful and thus reduce the all too frequent negative lumbar disk exploration.

The exact technic of handling the cysts still is open to question. Excision of the entire cyst, reported in a few cases of solitary cysts, is seldom possible, since extensive damage may be inflicted upon the numerous neurofibrils present in the cyst walls. Incision and imbrication with a fine silk suture is the method used by the author, but even with the utmost care the motor and sensory sacral nerve deficit was aggravated in Case 2.

One note of caution in the surgical exploration of the sacral canal may be worthy of record: it has been the author's experience that the acute flexion position, over rolls, etc., so commonly used for disk surgery is inadvisable when exploring the sacral canal. This position causes acute pressure on the abdomen and increases the intraspinal venous pressure to the point that venous distention appears with the inevitable troublesome venous hemorrhage. This is easily prevented by avoiding any pressure on the abdomen and thus making the exploration a relatively simple procedure.

SUMMARY

Perineurial cysts are another, though as yet uncommon, cause of low backache and

sciatic pain and in some instances of genital, sensory and motor symptoms. Diagnosis may be made by myelographic visualization of the cysts (in which pantopaque enters the cysts), roentgenographic evidence of erosion or hollowing-out of the sacral canal and, in some cases, only by surgical exploration of the sacral canal after negative explorations for lumbar disk protrusion (though occasionally a cyst may be an incidental finding).

Surgical management is directed toward collapsing the cyst (incision and imbrication) or excising the single cyst, if possible.

THREE ADDITIONAL CASE REPORTS

Case 6. A 58-year-old housewife had complained of progressive low back, left buttocks, perineum and sciatic pain over a 4-month period. Recently there had been weakness in the left lower extremity with sensory loss; both proved to be in the left sacral segments 2, 3 and 4.

The general physical examination failed to disclose any cause for these neurologic symptoms and signs. Repeated roentgenograms of the pelvis suggested slight erosion in the left half of the sacrum. A pantopaque myelogram was essentially normal. Surgical exploration of the sacrum disclosed a perineurial sacral cyst of the left S-3 root. This lesion was not opened. Further exploration disclosed an abscess occupying the bodies of S-2, 3 and 4 vertebrae. The abscess was evacuated and drained. The patient had considerable relief of pain but recently proved to have metastatic malignancy. It is believed that this cyst was only an incidental finding and of no definite clinical significance.

Case 7. A 38-year-old railroad brakeman fell on his back on March 28, 1955 and developed severe low back pain 30 minutes later. He was treated by conservative means for an acute low back injury but failed to improve. On June 16, 1955, he presented the usual signs of a lower lumbar protruded disk without demonstrable sensory changes. Percussion over the lower lumbar spine and the sacrum elicited severe radiating pain. A myelogram disclosed a small L-4 left defect and an S-2 level perineurial sacral cyst type of *sac defect*.

On June 23, 1955, a small L-4 disk was excised, and a perineurial sacral cyst measuring 2 x 3 x 1½ cm was found. This cyst appeared to be on the left S-4 root. It had enlarged moderately the proximal half of the sacral canal. There also was an enlargement of the left S-1 root suggestive of early cyst formation in it.

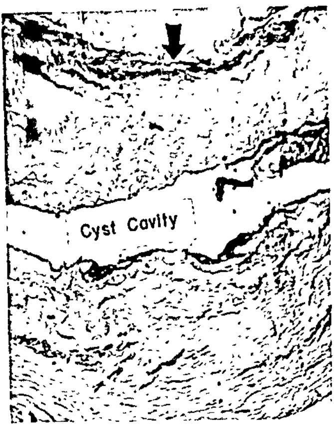


FIG. 8. Photomicrograph (Mallory trichrome × 175) of perineurial sacral cyst wall disclosing myelinated nerve fibers (arrow) in a fairly dense fibrous tissue.

The large perineurial sacral cyst was removed subtotally with some difficulties. The many nerve fibers in it were carefully dissected off the cyst wall, and a section of the wall was removed, followed by an imbricating type of suture (6-0 silk).

In spite of the gentleness used in handling these nerves, the postoperative course has not been a happy one. At first, the patient had a moderately severe sensory loss over the S-2, 3 and 4 dermatomes without loss of sphincter control, but with loss of power of erection. This has improved slowly until his sexual power has returned, along with about ¾ of his sensory function. Considerable low back and bilateral sciatic pain persisted for months until he was tried on Meticorten (prednisone), when the pain abated.

Case 8. A 58-year-old female was seen on July 8, 1955, with complaints of low back, left groin and left sciatic pain that began in February, 1955. In addition to the usual signs of an acute low back syndrome, the Lasègue sign was negative, and percussion over the sacrum elicited distress in the lower back and the left posterior thigh. A pantopaque myelogram disclosed a large perineural sacral cyst that on surgical exploration proved to measure 3.5 x 2.5 x 3 cm. in its

greatest diameters. It appeared to involve the left S-2 and S-3 roots. Sections of the cyst wall stained by H & E, Mallory's trichrome and Bodian stains disclosed myelinated nerve fibers coursing through the fibrous tissue of the cyst wall (Fig. 8).

TWO RECENT REPORTS IN THE LITERATURE

Two contributions appeared almost simultaneously in the literature recently. They are of considerable interest:

Seaman, William B., and Furlow, Leonard T.: The myelographic appearance of sacral cysts, J. Neurosurg. 13:88-94, 1956.

Schurr, Peter H.: Sacral extradural cyst: an uncommon cause of low back pain, J. Bone & Joint Surg. 37-B:601-605, 1955.

In the first, roentgenographic findings are stressed, but no attempt is made to differentiate between extradural cysts and perineurial sacral cysts, such as Schurr has emphasized. It might be shown further that it is not always easy to differentiate between the two in the sacral region. This may be the reason why Baker and Webb have used the noncommittal term *intrasacral meningocele.* Possibly this would have been a better description in Case 5, that of Dr. H E LeFever. Spinal extradural cysts elsewhere in the spinal column are an entirely different problem, as I have shown previously.[4]

REFERENCES

1. Browder, J.: See Tarlov, I. M. (Item 17).
2. Cloward, R. B.. Spinal extradural cysts, Ann. Surg. 105:401-407, 1937.
3. Cloward, R. B., and Bucy, P. C.: Spinal extradural cyst and kyphosis dorsalis juvenilis, Am. J. Roentgenol. 38:681-706, 1937.
4. Good, C. Allen, Adson, Alfred W., and Abbott, Kenneth H. Spinal extradural cyst (diverticulum of spinal arachnoid), Am J. Roentgenol 52:53-56, 1944
5. Jacobs, Lewis G ; Smith, James K., and Van Horn, Philip S.: Myelographic demonstration of cysts of spinal membranes, Radiology 62:215-221, 1954
6. Mérei, F. T Mit klinischen Symptomen einhergehende Zysten der Caudawurzein, Zentralbl Neurochir 13:212-218, 1953.
7. Rexed, B · Arachnoidal proliferations with cyst formation in human spinal nerve roots at their entry into the intervertebral foramina; a preliminary report, J. Neurosurg. 4:414-421, 1947.
8. Schlesinger, H.: Beiträge zur Klinik der Rückenmarks und Wirbeltumoren, Jena, G. Fischer, 1898.
9. Schreiber, F., and Haddad, B.: Lumbar and sacral cysts causing pain, J. Neurosurg. 8:504-509, 1951.
10. Strully, Kenneth J., and Heiser, Saul: Lumbar and sacral cysts of meningeal origin, Radiology 62:544-549, 1954.
11. Taheri, Z. E., Riemenschneider, P., and Ecker, A.: Myelographic diagnosis of sacral perineurial cyst, J. Neurosurg. 9:93-95, 1952.
12. Tarlov, I. M.: Cysts (perineurial) of the sacral roots; another cause (removable) of sciatic pain, J.A.M.A. 138:740-744, 1948.
13. ———: Perineurial cysts of the spinal nerve roots, Arch. Neurol. & Psychiat. 40: 1067-1074, 1938.
14. ———: Personal communication.
15. ———: Sacral Nerve-Root Cysts. Another Cause of the Sciatic or Cauda Equina Syndrome. Springfield, Ill., Thomas, 1953.
16. ———: Sacral nerve root cysts; clinical significance and pathogenesis, Arch. Neurol. & Psychiat. 68:94-108, 1952.
17. ———: Sacral nerve-root cysts; pathogenesis and clinical significance, J. Nerv. & Ment. Dis. 117:156-157, 1953.
18. Weiford, E. C.: Sacral perineurial cysts with a case report, Cleveland Clin. Quart. 17:106-111, 1950

Cystes Perineurial Revista del Litteratura e Reporto de Octo Casos

Summario in Interlingua

Cystes meningeal intraspinal esseva originalmente describite in 1898 per Schlesinger. In 1938 un specific typo de tal cystes esseva describite per Tarlov sub le nomine "cyste sacral perineurial," e dece annos plus tarde isto esseva inculpate como factor etiologic in le production de dolores infero-dorsal e sciatic. Le mesme conclusion se trovava in reportos per Rexed (1947), Weiford (1950), Schreiber e Haddad (1951), e in nove reportos per Tarlov in 1952 e 1953, como etiam in reportos per Strully e Heiser, Jacobs

et al., e Merci. A iste gruppo le presente autor adde un serie de octo casos. Il ha essite demonstrate que le majoritate de iste lesiones pote esser diagnosticate per medios radiographic. Tamen, il es probabile que il occurre un certe numero de cystes sacral perineurial que non es penetrate per Pantopaque sed con que fluido cerebrospinal se trova in communication.* Roentgenogrammas del sacro pote esser diagnostic si erosion e allargamento del canal sacral (area "cavate" de Browder) es evidente insimul con marcate attenuation del arco posterior del sacro.

Tarlov ha presentate datos in supporto del conception que iste cystes ha un etiologia traumatic, que illos es probabilemente le consequentia de hemorrhagia a in le radice nerval, o ab sanguine subarachnoide spinal o ab un trauma directe del radice. Le hemorrhagia se extende a un certe grado intra le spatio perineurial e postea distende e rumpe venas intra le radice e le ganglion nerval o se infiltra a in le structura directemente. Le absorption del hemorrhagia e del destruite texito nerval resulta in le formation del cystes.

Es sublineate le facto que le cystes extradural in le canal dorsal, lumbar, o sacral ha apparentemente nulle relation al cystes perineurial. Nos etiam signala que le cystes traumatic que occurre in le region lumbar in consequentia de operationes super discos intervertebral non debe esser confundite con le lesion hic considerate. Le duo lesiones es histologicamente facile a identificar.

Le symptomas e signos de quasi omne le reportate casos de cyste sacral perineurial—includente le casos hic reportate—esseva illos del syndrome sciatic e similissime al symptomas e signos del syndrome de herniate discos intervertebral. In alicun casos, incontinentia o retenentia urinari esseva presente insimul con le dolor. Un disturbation del sensation perineal esseva characteristic de circa un quarto del patientes. Symptomas reflexe e motor es etiam mentionate.

Le presente autor describe octo cystes perineurial intrasacral, quatro in masculos e quatro in femininas. Le etates del patientes variava ab 38 a 76 annos. Le configuration del cystes variava ab le occurrentia de un parve cyste super un radice sacral al presentia de multiple cystes (probabilemente sex) in distribution bilateral super le mesme numero de radices sacral. Un cyste esseva discoperite durante un operation chirurgic pro un abscesso in le corpores del sacro, probabilemente debite a un malignitate metastatic. Omne le altere patientes in nostre serie habeva essite subjicite a explorationes chirurgic con resultatos ab bon a dissatisfacente.

Le presente autor opina que le cystes sacral perineurial pote esser symptomatic e le causa del syndrome sciatic. Illos etiam pote esser discopertas incidental que non produce ulle symptoma del toto. Un situation analoge se recognosce communmente in connexion con le problema del protrundente disco intervertebral: Discos protrundente, e cystes similemente, non es necessarimente productive de symptomas. Si o non symptomas es manifeste depende probabilemente de plure factores. Istos include mechanic effectos local del cyste e progressive alterationes irritative in le radice sensori posterior, includente neuritis ischemic.

Le presente autor opina que le lesion hic discutite merita le plus serie consideration. Isto es specialmente ver in casos de obscur syndromes sciatic.

* Si Pantopaque remane presente in le canal spinal, il es a recommendar repeter le examine radiographic le dies sequente, proque a vices tal cystes se plena del oleo e assi deveni demonstrabile. Isto occurreva in un del casos reportate in le presente serie.

greatest diameters. It appeared to involve the left S-2 and S-3 roots. Sections of the cyst wall stained by H & E, Mallory's trichrome and Bodian stains disclosed myelinated nerve fibers coursing through the fibrous tissue of the cyst wall (Fig. 8).

TWO RECENT REPORTS IN THE LITERATURE

Two contributions appeared almost simultaneously in the literature recently. They are of considerable interest:

Seaman, William B., and Furlow, Leonard T.: The myelographic appearance of sacral cysts, J. Neurosurg. 13:88-94, 1956.

Schurr, Peter H.: Sacral extradural cyst: an uncommon cause of low back pain, J. Bone & Joint Surg. 37-B:601-605, 1955.

In the first, roentgenographic findings are stressed, but no attempt is made to differentiate between extradural cysts and perineurial sacral cysts, such as Schurr has emphasized. It might be shown further that it is not always easy to differentiate between the two in the sacral region. This may be the reason why Baker and Webb have used the noncommittal term *intrasacral meningocele*. Possibly this would have been a better description in Case 5, that of Dr. H. E. LeFever. Spinal extradural cysts elsewhere in the spinal column are an entirely different problem, as I have shown previously.[4]

REFERENCES

1. Browder, J.: See Tarlov, I. M. (Item 17).
2. Cloward, R. B.: Spinal extradural cysts, Ann. Surg. 105:401-407, 1937.
3. Cloward, R. B., and Bucy, P. C.: Spinal extradural cyst and kyphosis dorsalis juvenilis, Am. J. Roentgenol. 38:681-706, 1937.
4. Good, C Allen, Adson, Alfred W., and Abbott, Kenneth H.: Spinal extradural cyst (diverticulum of spinal arachnoid), Am. J. Roentgenol. 52:53-56, 1944.
5. Jacobs, Lewis G.; Smith, James K., and Van Horn, Philip S.: Myelographic demonstration of cysts of spinal membranes, Radiology 62:215-221, 1954.
6. Mérei, F T . Mit klinischen Symptomen einhergehende Zysten der Caudawurzein, Zentralbl. Neurochir 13:212-218, 1953.
7. Rexed, B. Arachnoidal proliferations with cyst formation in human spinal nerve roots at their entry into the intervertebral foramina; a preliminary report, J. Neurosurg. 4:414-421, 1947.
8. Schlesinger, H.: Beiträge zur Klinik der Rückenmarks und Wirbeltumoren, Jena, G. Fischer, 1898.
9. Schreiber, F., and Haddad, B.: Lumbar and sacral cysts causing pain, J. Neurosurg. 8:504-509, 1951.
10. Strully, Kenneth J., and Heiser, Saul: Lumbar and sacral cysts of meningeal origin, Radiology 62:544-549, 1954.
11. Taheri, Z. E., Riemenschneider, P., and Ecker, A.: Myelographic diagnosis of sacral perineurial cyst, J. Neurosurg. 9:93-95, 1952.
12. Tarlov, I. M.: Cysts (perineurial) of the sacral roots; another cause (removable) of sciatic pain, J.A.M.A. 138:740-744, 1948.
13. ———: Perineurial cysts of the spinal nerve roots, Arch. Neurol. & Psychiat. 40: 1067-1074, 1938.
14. ———: Personal communication
15. ———: Sacral Nerve-Root Cysts. Another Cause of the Sciatic or Cauda Equina Syndrome. Springfield, Ill., Thomas, 1953.
16. ———: Sacral nerve root cysts; clinical significance and pathogenesis, Arch. Neurol. & Psychiat. 68:94-108, 1952.
17. ———: Sacral nerve-root cysts, pathogenesis and clinical significance, J. Nerv. & Ment. Dis. 117:156-157, 1953.
18. Weiford, E. C.: Sacral perineurial cysts with a case report, Cleveland Clin. Quart. 17:106-111, 1950.

Cystes Perineurial Revista del Litteratura e Reporto de Octo Casos

Summario in Interlingua

Cystes meningeal intraspinal esseva originalmente describite in 1898 per Schlesinger. In 1938 un specific typo de tal cystes esseva describite per Tarlov sub le nomine "cyste sacral perineurial," e dece annos plus tarde isto esseva inculpate como factor etiologic in le production de dolores infero-dorsal e sciatic. Le mesme conclusion se trovava in reportos per Rexed (1947), Weiford (1950), Schreiber e Haddad (1951), e in nove reportos per Tarlov in 1952 e 1953, como etiam in reportos per Strully e Heiser, Jacobs

et al., e Merci. A iste gruppo le presente autor adde un serie de octo casos. Il ha essite demonstrate que le majoritate de iste lesiones pote esser diagnosticate per medios radiographic. Tamen, il es probabile que il occurre un certe numero de cystes sacral perineurial que non es penetrate per Pantopaque sed con que fluido cerebrospinal se trova in communication.* Roentgenogrammas del sacro pote esser diagnostic si erosion e allargamento del canal sacral (area "cavate" de Browder) es evidente insimul con marcate attenuation del arco posterior del sacro.

Tarlov ha presentate datos in supporto del conception que iste cystes ha un etiologia traumatic, que illos es probabilemente le consequentia de hemorrhagia a in le radice nerval, o ab sanguine subarachnoide spinal o ab un trauma directe del radice. Le hemorrhagia se extende a un certe grado intra le spatio perineurial e postea distende e rumpe venas intra le radice e le ganglion nerval o se infiltra a in le structura directemente. Le absorption del hemorrhagia e del destruite texito nerval resulta in le formation del cystes.

Es sublineate le facto que le cystes extradural in le canal dorsal, lumbar, o sacral ha apparentemente nulle relation al cystes perineurial. Nos etiam signala que le cystes traumatic que occurre in le region lumbar in consequentia de operationes super discos intervertebral non debe esser confundite con le lesion hic considerate. Le duo lesiones es histologicamente facile a identificar.

Le symptomas e signos de quasi omne le reportate casos de cyste sacral perineurial—includente le casos hic reportate—esseva illos del syndrome sciatic e similissime al symptomas e signos del syndrome de herniate discos intervertebral. In alicun casos, incontinentia o retenentia urinari esseva presente insimul con le dolor. Un disturbation del sensation perineal esseva characteristic de circa un quarto del patientes. Symptomas reflexe e motor es etiam mentionate.

Le presente autor describe octo cystes perineurial intrasacral, quatro in masculos e quatro in femininas. Le etates del patientes variava ab 38 a 76 annos. Le configuration del cystes variava ab le occurrentia de un parve cyste super un radice sacral al presentia de multiple cystes (probabilemente sex) in distribution bilateral super le mesme numero de radices sacral. Un cyste esseva discoperite durante un operation chirurgic pro un abscesso in le corpores del sacro, probabilemente debite a un malignitate metastatic. Omne le altere patientes in nostre serie habeva essite subjicite a explorationes chirurgic con resultatos ab bon a dissatisfacente.

Le presente autor opina que le cystes sacral perineurial pote esser symptomatic e le causa del syndrome sciatic. Illos etiam pote esser discopertas incidental que non produce ulle symptoma del toto. Un situation analoge se recognosce communmente in connexion con le problema del protrundente disco intervertebral: Discos protrundente, e cystes similemente, non es *necessarimente* productive de symptomas. Si o non symptomas es manifeste depende probabilemente de plure factores. Istos include mechanic effectos local del cyste e progressive alterationes irritative in le radice sensori posterior, includente neuritis ischemic.

Le presente autor opina que le lesion hic discutite merita le plus serie consideration. Isto es specialmente ver in casos de obscur syndromes sciatic.

* Si Pantopaque remane presente in le canal spinal, il es a recommendar *repeter le examine* radiographic le dies sequente, proque a vices tal cystes se plena del oleo e assi deveni demonstrabile Isto occurreva in un del casos reportate in le presente serie.

Section II

GENERAL ORTHOPAEDICS

16

Experiences with Congenital Scoliosis

HENRY F. ULLRICH, M.D.*

Fortunately, congenital scoliosis and other serious congenital abnormalities of the spine are not too common. The arrival of a child into a family with a congenitally deformed spine may initiate a train of unhappy and trying experiences for the family, the patient, the orthopaedist, the pediatrician and the hospital. A normally expectant father and mother will have their lives changed, and, unless they develop insight into the problem, these changes at times are often profound and irreversible. If there has been any serious abnormality on either side of the family, familial complications arise and sometimes are not too easily settled by anyone.

Whatever the etiologic factor or factors may be—whether the congenital deformity represents some error of differentiation, some hereditary defect or some other influence acting upon a normally fertilized ovum—after the arrival of the baby they are inconsequential. The fact remains that one is dealing with a process that is not completely rectifiable in any case, and in some the deformity is, or will be, so severe and hopeless that treatment resolves itself into mere palliation. Various theories regarding etiology have been advanced, but they do not explain all cases and are, indeed, difficult to correlate. Hereditary theories do not explain all, nor do the mechanistic theories, in which various pressures are exerted abnormally. The recent works of Duraswami are most interesting and may explain at least some of these abnormalities. These abnormalities are so bizarre, do not follow any fixed pattern, and for this reason, perhaps, other factors may be involved in the production of these errors of development.

The pattern that one observes in so-called idiopathic scoliosis, namely, that of a right dorsal left lumbar curve in a preadolescent, does not fit into this pattern. Furthermore, congenital abnormalities of the spine do not exist usually as isolated deformities; often they are associated with dislocated hips, arthrogryphosis, clubfeet, loss of bowel and bladder control and, not uncommonly, flaccid paralysis of lower extremities with or without anesthesia. Of 3,266 admissions to Kernan Hospital, Baltimore, Md., 12 cases of congenital scoliosis had serious enough disability or deformity to warrant serious consideration of the curve itself, and there were other abnormalities. Of these, only 4 were of such a nature as to warrant consideration of partial correction of the curve and realignment of the trunk.

According to Kuhns,[4] the incidence of congenital scoliosis is 11 per cent as compared with the idiopathic scolioses; according to Kleinberg,[3] 5 per cent; and, according to Cobb,[1] 2 per cent. Of this series, 4 per cent were found to be due to congenital scolioses. Children admitted primarily for treatment of clubfeet were x-rayed routinely for spinal abnormalities, but of these no serious deformity was found sufficient to warrant specific treatment for the curve itself.

* Baltimore, Md

Age of Discovery	
At birth	2
3 months	1
9 months	1
18 months	3
2 years	1
3 years	2
3½ years	1
7 years	1

Of these patients, 4 were males and 8 were females. Rib abnormalities were involved in those in the thoracic area. The operations performed were: calcaneal tendon lengthenings on 8 feet of 4 patients; spinal fusion in 4 patients, with a total of 7 spinal fusion operations; spinal explorations by neurosurgeons in 3 patients; plaster-cast immobilization and correction in 1 patient; epiphysial stapling of tibia, for shortening, in 1.

Concerning the hospital stay, the time varied from 8 days to 720 days. The stay of 7 patients was longer than 150 days.

Mental changes were not observed in the form of any degree of deficiency; in fact, in 2 children it was found that they were exceptionally bright and probably even a bit precocious. This may be attributed possibly to their having been in hospital for a long period or having been nurtured more carefully by adults because of their handicap and just appeared to be brighter. At any rate, they were extremely happy children who were most attractive and well adjusted. The child who spent 720 days in hospital was transferred to a state hospital for chronic illness and was depressed to a severe degree. This is understandable because of the long period of hospitalization, with little or nothing to show for it regarding ultimate function or rehabilitation.

FAMILY HISTORY

Five of the 12 patients came from homes that were not ideal In 1 of them, there was existing deformity of parents; in another, the parents were alcoholic, in 2 more the parents were separated; and in the last the parents were not known and the child was presumed to be illegitimate.

CLINICAL APPEARANCE

The type of patient in whom hemivertebra seems to be the chief problem presents the clinical appearance of one in whom a curvature exists but rotation seems to be out of all proportion to the angularity of the clinical appearance of the curve. In the flank a hard mass is observed. This is the rotated vertebral body. On lateral bending it does not recede or disappear as one would expect it to do in the case of an acquired scoliosis. The rib prominence in the thoracic area is usually extremely severe, sharp and angulated, and the kyphosis is much more severe than what one would expect in an acquired curvature of the same degree of angularity. There is also extreme rigidity, and with such rigidity of the curvature in the thoracic area one should be led to suspect that a congenital abnormality exists When the thoracic curvature extends into the lumbar area and there are insufficient vertebrae to initiate a compensatory curve in the opposite direction, a trunk shift sets in early and is followed by increase in trunk shift and displacement or decompensation. This continues at times until the patient is entirely out of balance. The inability of the lumbar spine to compensate is the result of the lumbar spine's being less flexible than normal, or there may also be some existing congenital abnormality in the lumbar spine. This precludes the possibility of the formation of a reverse compensatory curve In addition to this, a tuft of hair in the lumbar area or in the sacrum always should lead one to suspect that probably a congenital abnormality exists.

The roentgenograms which are taken are self-explanatory, and, as in other scolioses, they always reveal much more angularity of the curve or rib abnormalities than one would suspect from clinical examination, no matter how carefully done. Bending roentgenograms to the right and to the left reveal that there often is little or no flexibility at

the area of greatest angularity. These films are studied to determine the degree of flexibility of the more uninvolved portions of the spine. Only at the ends of the curve is one apt to detect any degree of flexibility. In such cases, if there is any degree of flexibility at the ends of the curve, it may prove to be of great help in considering the rebalancing of the trunk at a time elected for more radical therapy.

The 4 patients who underwent surgery were about 6 years of age, when it was felt that there had been sufficient bony development, as well as somewhat better co-ordination, of the patient. It was felt also that, at the age of 6, the operative risk would be much less than if the child were younger. Anesthesia was not too difficult; all were intubated before surgery in order to establish a free airway. In those with Risser jackets, one had to be sure to have the head in enough extension to allow for less traumatic insertion of an endotracheal tube. Although the amount of hemorrhage was not excessive, it was somewhat more profuse than what one would have expected in operating upon a spine that had been curved for causes other than congenital. At operation, bony landmarks were found to be distorted and were consistent with the roentgen findings. At times it was difficult to identify and locate accurately the marker on a vertebral body. It was somewhat more difficult than in other spines to retract and separate subperiosteally the soft tissues. In those instances in which there were bifid laminae, extreme caution had to be exercised in order to prevent inadvertent nicking or opening of the dura. This happened in 1 case, and some delay occurred in order to close the leak in the dura before the operation could be continued. This unnecessary delay probably could have been prevented by more careful separation of the overlying soft tissues.

The extreme rotation presented some difficulty at times, and transverse processes could be mistaken without too much difficulty for spinous processes In exposing these con-

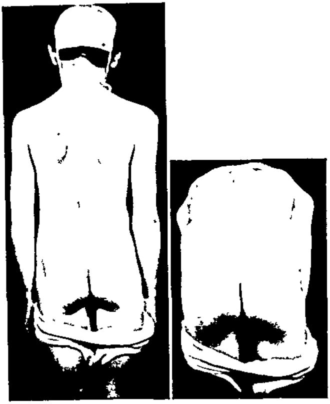


FIG. 1. Clinical appearance: hemivertebra L-2. Note left flank fullness and more apparent rotation than what one would expect for the angularity of the curve.

genitally scoliotic spines, one will encounter at times spontaneously or naturally fused laminae. Little or no time need be spent here; time can be spent more profitably on other joints where this is the case. At the time of turning of the chips, the fused laminae here also were scarified by use of a sharp gouge.

In those cases in which neurosurgical exploration was done, the findings were those of a thinned dura and nerve root densely matted up in scar. The neurosurgical exploration had little or no demonstrable effect on the ultimate outlook or outcome of any patient. However, it did establish, once and for all, exact knowledge of the existing pathologic changes in the cord, the dura and the nerve roots, and, so far as was determined, these changes were irreversible. Possibly decompression may have been of some help, but not too much.

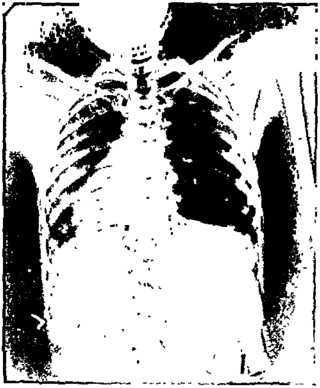


FIG. 2. Roentgen appearance of Figure 1.



FIG. 3. Clinical appearance.



FIG. 4 *(Left)* Preoperative roentgenogram. *(Right)* Immediate result.

CASE HISTORIES

Case 1 (Figs. 1 & 2). This deformity was found to be due to hemivertebra. Because of good trunk alignment, this curve was fused *in situ* in an effort to slow down or arrest the progress of the deformity. Here it was found that the scoliosis extended well down into the lumbar spine, and, because of this, it was felt that a hindering force was necessary in the form of a fusion in order to prevent further decompensation. There were not enough joints below the hemivertebra to allow opposite curvature to initiate compensation, and, therefore, increase in angularity of this curve due to hemivertebra would be accompanied by considerable decompensation and marked hip asymmetry. Fusion was done from the 1st to the 4th lumbar vertebrae. At operation, hemilamina was encountered, but fusion was not done across this. The patient spent 36 weeks in plaster-cast immobilization, after which she was allowed to be free without any additional support. No attempt was made at resection of vertebral bodies because the clinical alignment was good. It was felt that this probably would not be worth the risk in an asymptomatic spine, and that the simpler procedure of fusion of the posterior elements was sufficient.

Case 2 (Figs. 3-5). This child came under observation at the age of 5, and was found to have multiple congenital abnormalities. In addition, there was decompensation of the trunk off to the left, and it was hoped that, by increas-



FIG. 5. Result 3 years after operation.

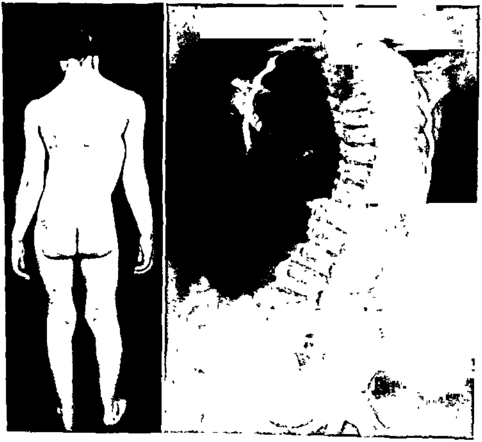


FIG 6. *(Left)* Clinical appearance. *(Right)* Preoperative roentgenogram.

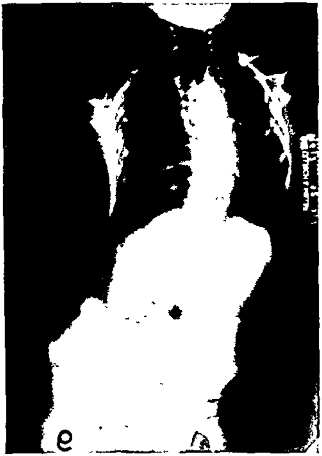


FIG. 7. Postoperative roentgenogram. Note improvement in balance.

ing the lumbar curve and fusing into the lumbar spine, a secondary curve, as a result of the bending of the trunk to the left, would increase the angularity of the lumbar curve and thereby decrease the child's trunk shift. This correction was maintained in spite of his growth, and fusion was done in a Risser type jacket. The postoperative course was uneventful. At operation it was found that there were no laminae at the 10th, the 11th or the 12th thoracic levels. This defect, after being dissected carefully, was reinforced with bank bone in order to bridge this huge gap.

Case 3 (Figs. 6 & 7). This child presented a similar problem with regard to previous deformity. This was progressive, and it was noticed at the age of 2. She was admitted to the hospital at the age of 5 years and 9 months, and a similar type of correction was done. On examination, it was found that there was a rather large tuft of hair in the lumbar area, and similar reasoning was carried out in the case of this child.

Case 4 (Figs. 8 & 9). This child was given benefit of myelographic studies and found to be *normal. Fusion was necessary, to be done in* 3 stages, but was preceded by rib resection on the concave side of the curve. This was carried out in an effort to facilitate further correction. It did not allow any material change in the correctibil-

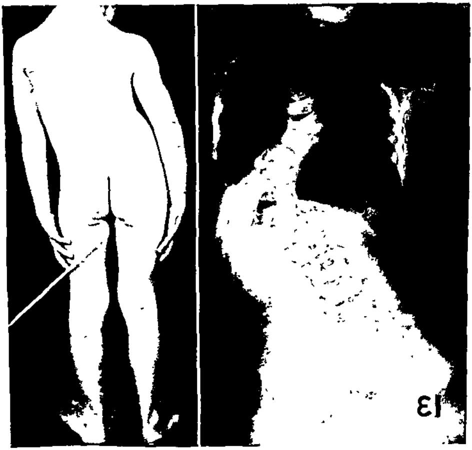


FIG. 8. *(Left)* Clinical appearance. *(Right)* Preoperative roentgenogram

ity of the patient's thoracic curve. However, she got along reasonably well following operation, and her general trunk alignment remained satisfactory.

Other defects were treated either while the spine was being cared for or afterward. The feet that required care were given either braces or tendo achillis lengthening in order to permit the wearing of shoes or to decrease the equinus deformity as much as possible for weight-bearing. In those in whom there was some sensory loss, it was found necessary to pay particular attention to the skin to minimize the formation of pressure areas and decubiti. In 1 case, epiphysial stapling of a tibia and a fibula was done in the hope of decreasing the discrepancy in length. Decubiti were cared for by routine toilet of the ulcer and by relieving prominences, and they healed uniformly.

Incontinence was not too much of a problem at first. The entrance of an incontinent child into school makes him extremely self-conscious and, in fact, a social outcast so far as other children are concerned, even though these same children are handicapped in other ways. In 1 instance a child had treatment for urinary incontinence at another hospital by transplantation of the ureters into the sigmoid. This converted the anus into cloaca and at least took care of the urinary incontinence. This helped somewhat, but in spite of it there was still incontinence of bowels, and strict dietary regulation was required. Perhaps, in selected cases, a looping of the anal outlet, as outlined by Pickrell, may offer some hope for these unfortunate incontinent individuals.

None of these children had any direct attack on the vertebral bodies themselves. The object was to bend the trunk against the convexity of the more serious curve in an



Fig 9. (*Left*) Roentgenogram and (*right*) clinical appearance 3 years after operation.

attempt to rebalance the trunk and compensate and maintain an otherwise decompensated scoliosis. By this procedure it was hoped that, as shown in these several children, the entire treatment would be worth while in partly re-establishing trunk balance. The results on these patients showed that immediately after surgery there was no serious shock. Transfusion was given during and after operation. There were no deaths, and no infections followed surgery. They were kept in bed routinely 12 weeks after surgery, during which time they were allowed to move from one side to the other as their condition permitted. During convalescence they managed to keep up their studies in school, and got along well.

This would not be complete without a word regarding the severe hardship inflicted on parents and others that is equaled by few congenital abnormalities. With an incontinent child, to say nothing of his being deformed, at or near school age, it is little wonder that there are apt to be familial difficulties, and the homes of these children are not always ideal.

To summarize: In the mild cases that are well balanced, no specific treatment of the spine itself is required. Observation is necessary from time to time to determine if there is any tendency toward decompensation. Some few can be rebalanced safely, in the hope of restoring at least some degree of the lost balance due to trunk shift. Treatment is long, and sometimes the ultimate result does not seem to be too satisfactory, but it was felt in these cases that it was quite worth while, although there was not the dramatic improvement that one often obtains in idiopathic or acquired scolioses. It would seem to be sufficiently worth while to try the general plan in an effort to rehabilitate these children for as useful a life as is possible without too great a risk endeavoring to attack directly the congenitally deformed vertebral body or bodies.

REFERENCES

1. Cobb, J. A.: Outline for Study of Scoliosis, Am. Acad. Orthop. Surgeons Instructional Course Lectures, vol. V, p. 276, 1948.
2. Duraswami, P. K.: J. Bone & Joint Surg. 34B:661, 1952.
3. Kleinberg, S.: Scoliosis, Pathology, Etiology and Treatment, p. 107, Baltimore, Williams & Wilkins, 1951.
4. Kuhns, J. G.: Congenital scoliosis, New England, J. Med. 210:1310, 1934.

Experientias con Scoliosis Congenite

Summario in Interlingua

Iste reporto se occupa del patientes qui esseva presentate pro tractamento de scoliosis congenite al Hospital James Lawrence Kernan ab 1936 a 1953. Le serie total consiste de 12 patientes. Ab istes, quatro habeva scoliosis de grado sufficiente pro suggerer alineamento o re-alineamento del trunco Nulle o pauc correction genuin del curvatura per se esseva obtenite. Le uso de corsets a correction con subsequente fusion spinal valeva le pena in iste quatro casos. Leve curvas e curvas auto-compensatori requireva nulle tractamento. Exploration neurochirurgic establiva le stato del subjacente alterationes de cordon e radice sed non influentiava le prospectos final.

17

Studies of the Use of Cultured Calf Bone in Human Bone Grafts

E. J. TUCKER, M.D., F.A.C.S.*

The usefulness of heterogenous bone in human beings has been of interest to orthopaedic surgeons for many years, often because of its unlimited supply. Since the bone bank has become such a necessary adjunct to orthopaedic surgery, more interest now is present in this material for several other well-recognized reasons: (1) orthopaedic procedures are greatly simplified; (2) this bone is available at all times; (3) there is freedom from danger of syphilis and from a hepatitis virus contamination; (4) the material is obtained immediately after death; (5) there is no legal question involved in obtaining the bone.

Recently a group of able attorneys conducted a legal research in regard to human tissue banks, and their summary was as follows:

> In conclusion, there would appear to us no adequate solution to your problem of the legality of human tissue banks; at least insofar as the laws of our state are concerned. Although it is our understanding that bones and certain organs are removed frequently from bodies by members of the medical profession for transplantation, in our opinion it is highly questionable that they are doing so within their legal rights, even though the consent of the nearest of kin is obtained.†

The principal objection to the use of animal grafts is their incompatibility with human beings. It is the purpose of this report to show that calf bone may be prepared in such a manner as to eliminate heterogenous bone reaction and also make this type of graft conducive to early union.

The term *cultured calf bone* is used because the bone is stored in the nutrient media of the same species, for purposes that are outlined in this report. This term also indicates that the tissue is subjected to numerous bacteriologic cultures before it reaches the operating room.

HISTORY OF HETEROGENOUS BONE GRAFTS

The earliest record of a successful bone transfer is one in which heterogenous bone was used. In 1668, van Meekeren[15] recorded an implant taken from a dog's skull and placed in a skull defect of a Russian soldier. This operation was a success, and healing was good; however, the graft later was removed by order of the Church.

In 1885, Ollier[9] reported some 60 cases of heterogenous grafts. Although the results of these cases were poor, Ollier did direct the attention of the medical world to the possibilities of this work. Then followed the reports of Schmitt,[12] in 1893, Barth,[2] in 1895, Axhausen,[1] in 1909, and Baschkirzew and Petrow,[3] in 1912. All of these were reports on the use of heterogenous bone grafts, the results of which were not satisfactory. There-

* Houston, Texas.

† Private investigation conducted by Andrews, Keith, Kurth and Campbell, Houston, Texas.



FIG. 1. Photomicrograph of a section of calf bone (× 125) which had been denuded of its periosteum and stored in bovine plasma for a period of 6 months at 40° F. Note the large number of bone cells present, the fibrous proliferation from the surface of the bone, and the loose structure of the bone itself.



FIG 2 Photomicrograph (× 125) of a section of calf bone after being stored at −10° F for 6 months This section came from the same bone as that in Figure 1, and shows relatively few bone cells, no proliferations from the surface of the bone, and a compact bone structure



FIG. 3. Photomicrograph (× 150) of calf bone stored in bovine plasma for 3 months, showing the formation of collagenous and elastic fibers out from the bone.



FIG. 4. Photograph of calf bone chips through a dissecting microscope (× 20). This bone had been stored in bovine plasma at 40° F for 3 months. Note the massing together of the chips with a dense fibrous formation.



FIG. 5. Photograph of calf bone chips from the same bone as that shown in Figure 3. These chips were frozen for a period of 3 months at −10° F. Note the absence of fibrous formation.

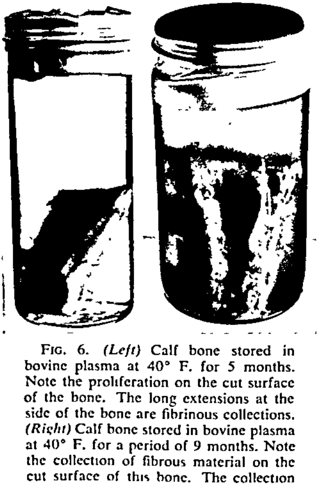


FIG. 6. *(Left)* Calf bone stored in bovine plasma at 40° F. for 5 months. Note the proliferation on the cut surface of the bone. The long extensions at the side of the bone are fibrinous collections. *(Right)* Calf bone stored in bovine plasma at 40° F. for a period of 9 months. Note the collection of fibrous material on the cut surface of this bone. The collection at the top and the sides of the bone is fibrinous deposit.

after, records of heterogenous grafts did not appear for some 25 years.

Orell,[11] in 1934, renewed the interest in this form of bone grafting when he published a report on the use of os purum. This report was one of the first scientific methods of preserving heterogenous bone for transplantation, and consisted chiefly of a physiochemical process of removing the fat and connective tissue from bone. It was used successfully in 4 cases of osteosynthesis. The same author,[10] in 1937, modified the use of os purum first by implanting small strips of the bone beneath the periosteum of the tibia. Later, this material was removed and transplanted to the desired place. Bone thus treated was known as *os novum*. Orell reported the successful use of os novum in 50 cases. Esnaurrizar[5] corroborated this work in 1940 by reporting the successful use of bone thus prepared in cases of tuberculous spondylitis, pseudarthrosis and an old fracture of the patella.

J. Judet, R. Judet and Arviset[8] should be credited with the first successful operation of a frozen calf-bone bank. In 1949, these authors showed that frozen calf bone could be used with results comparable with those obtained from frozen human bone. In 1953, Guilleminet, Stagnara and Perret[6] reported a



FIG. 7. Sections of human bone being placed on the metatarsal of a calf as onlay grafts. The segment of bone on the left is human bone, removed 6 hours post mortem and stored in human plasma for 5½ months. The segment of bone on the right is human bone that had been stored in human plasma for 15½ months.



FIG. 8. Section of frozen human bone on the left and a section of autogenous calf bone on the right, being placed on the opposite leg of the same calf as shown in Figure 7.

series of 80 cases in which frozen calf bone was used with only 10 per cent failures.

The most recent publication on calf-bone grafts is that of Terhune and Shannon,[13] who published a series of 27 major bone grafts using frozen calf bone with results comparable with those from autogenous bone. Approximately 20 per cent nonunions were reported in this series.

EXPERIMENTAL STUDIES

Effect of Temperature on Storage of Calf Bone

When calf bone is stored in bovine plasma at ordinary icebox temperatures (40° F.-45° F.), the bone cells will remain intact for long periods of time. Regardless of the discussion pro and con as to the viability of these cells, they are indisputably present. Figure 1 is a photomicrograph of a section of calf bone that was denuded of its periosteum and stored in bovine plasma at 40° F. for a period of 6 months. This section illustrates the large number of osteocytes present, a fibrous proliferation from the surface of the bone and a loose structure of the bone itself. Figure 2 is a section from the same piece of calf bone as Figure 1, stored at minus 10° F. for 6 months. In this section there are relatively few bone cells, there is no fibrous proliferation, and the bone structure is much more compact.

The unorganized proliferation, shown in Figure 1, also has been observed by Dobrowalskaja[4] and Haas.[7] The author[14] also has reported previously an extensive proliferation from bone when stored in the plasma of the same species. This formation consists chiefly of collagenous fibers and long coiled elastic fibers, as illustrated in Figure 3. Other illustrations of fibrous formations are shown in Figures 1, 4 and 6. This fibrous material is the ground substance, or apatite fibers, of early bone formations, and in all probability it is responsible for the lack of foreign body reactions and the rapid union of this type of bone graft.

Compatibility of Calf-Bone Implants with the Human Body

Sections of human bone which had been stored in human plasma at 40° F. for 5½ months and 15½ months were placed as onlay grafts on the first metatarsal of a calf (Fig. 7). In the opposite leg of the same animal, a section of frozen human bone and a



FIG. 9. Six weeks' postoperative photograph of human bone that had been stored in human plasma and then used as onlay graft of a calf's leg, as shown in Figure 7.



FIG. 10. Photomicrograph (× 120) through the heterogenous graft in Figure 9, showing union of the graft with no foreign body reaction. The bone on the left is calf bone, and the bone on the right is human bone.

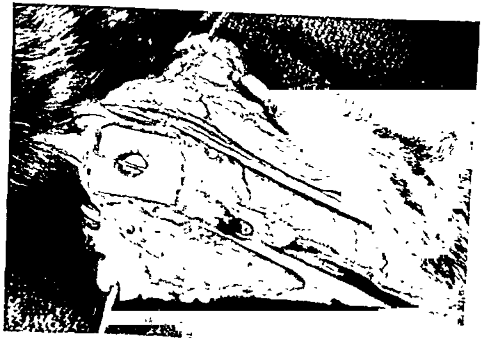


FIG. 11. Six weeks' postoperative photograph of the grafts in Figure 8, showing nonunion of the frozen human bone on the left and union of the autogenous bone on the right.

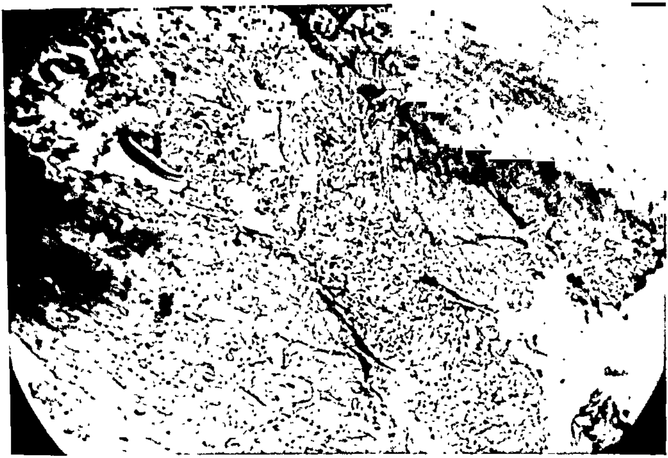


FIG. 12. Photomicrograph (× 125) through the onlay graft of frozen human bone on calf bone shown in Figure 11. There are nonunion of the graft and marked foreign body reaction between the frozen bone and the recipient.

section of the calf's own bone were used as control grafts.

Six weeks after operation there was union of the heterogenous grafts (Fig. 9). Microscopic section through the grafts showed no reaction between the human bone, which had been preserved in plasma, and calf bone (Fig 10). The frozen human bone in the opposite leg of the same animal showed no union after 6 weeks (Fig. 11). Section through the frozen human bone graft showed a marked foreign body reaction with fragmentation and absorption of the graft (Fig. 12). In the control graft of calf autogenous bone, there was early union, though not as marked as human bone, which had been preserved in plasma (Figs. 11 and 13).

The compatibility of human bone, preserved in plasma, to calf bone having been demonstrated, it was necessary to ascertain if calf bone preserved in bovine plasma was just as compatible with man. Accordingly, calf bone was preserved in bovine plasma for a period of 6 months at 40° F. This bone then was ground to a fine consistency under sterile conditions and placed in fresh tooth-extraction sockets of the maxilla of a human being. The implant was held in place by suturing the mucoperiosteal flap over the sockets. Following this procedure, there was no local reaction whatsoever round the bone, and the soft tissue healed with unusual rapidity. Eight weeks after implantation, there was true cancellous bone formation in the tooth sockets, as shown in Figure 14.

PRESERVATION OF GRAFTS AND TECHNIC OF THEIR USE

It was found that the density of cortical bone and the number of osteocytes per field in a 300-pound calf are about the same as those of a normal adult human being. How-



Fig. 13. Photomicrograph (× 230) of onlay graft of autogenous calf bone shown in Figure 11: early union and no foreign body reaction.



Fig. 14. Roentgenogram of tooth sockets of a human being 8 weeks after extraction and implantation of ground cultured calf bone. Note the true bone formation in these sockets. (E. G. Giles, D.D.S.)

ever, cancellous bone in an animal of this size is much less dense than in adult bone. The trabecular framework is much finer, and the spaces between the trabeculations are much larger.

The animal to be used in obtaining the bone is passed by government inspection and tested for tuberculosis and brucellosis. Immediately after the animal is killed, it is suspended by its hind feet and bled by severing the jugular vein, thus preventing any reflux through the portal system into the extremities. The leg to be used is shaved, scrubbed and removed proximal to the scapula or through the hip joint, as the case may be; then it is carried to the laboratory. Here it is scrubbed again with strong antiseptics and singed thoroughly with a flame. After a

final painting with tincture of iodine, it is wrapped in sterile sheets and carried into a sterile operating room.

The leg then is opened and sterile sheets are braded to the skin. The bone is lifted out under rigorous aseptic technic. Sections of cortical bone suitable for grafting are cut by means of a vise and a hand bone saw. The cancellous bone is ground into bone chips by means of a hand mill. All of the bone for implants is placed in 1 large container and covered with a solution of 20 per cent bovine plasma in physiologic saline. One million units of penicillin and 2 Gm. of



FIG. 15. *(Top)* Fashioning an "H" graft of cultured calf bone. Note that the graft is wrapped in gauze to protect the bone from the jaws of the vice. *(Bottom)* An "H" graft, after being fashioned to fit its bed, being kept in its plasma solution until the graft bed is ready to receive it. This graft is being prepared for the spinal fusion shown in Figures 16 to 22.

streptomycin is added to each liter of solution. The container is sealed with a sterile cap and placed in an ordinary refrigerator at 40° F. After the bone has been in storage for 14 days, a portion of the solution is removed for testing. Thus the bone is cultured with anaerobic, aerobic media and guinea-pig inoculation. When all cultures are proved to be negative, the bone is removed from the large container under strict asepsis and placed in individual containers with fresh plasma solution. A final culture then is made of each container with thioglycollate broth media.

Up to the present, in more than 150 cultures, 5 pieces of bone have been discarded because of contamination. The organism in each of these was an airborne fungus.

Sensitivity

Test serum for intradermal injections should accompany the graft, and the patient to receive the graft should be tested prior to operation. Mild to moderate sensitivity reactions may be disregarded. People strongly positive skin reactions woul receive this bone. The storage solution tains penicillin G and streptomycin; ther in people who are known to have an a to these drugs, the bone should b through several washings of normal at the operating table before it is used

TECHNIC OF THE USE OF THE BONE GRAFT

As in all bone grafts, a most rig aseptic technic is required, with the u antibiotics, postoperatively according t dividual indications. A healthy vascula for the graft is most necessary. All j fected cases should be selected most fully. In preparing the graft and the hand instruments, such as rongeurs an teotomes, should be used instead of m driven instruments. The graft should be ioned with a vise and a coping saw 15, *top*). The heat from motor-driven produces heat necrosis, and nonunion



Fig 16 An "H" graft of cultured calf bone being locked in position with good apposition to its surrounding bed. The spinous processes, above and below, were undercut to hold the graft in position.

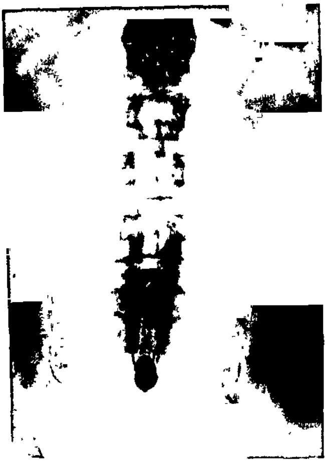


FIG. 17. Anteroposterior roentgenogram of the fusion shown in Figure 16. This was taken 4 weeks after operation, and shows the graft to be in good position.

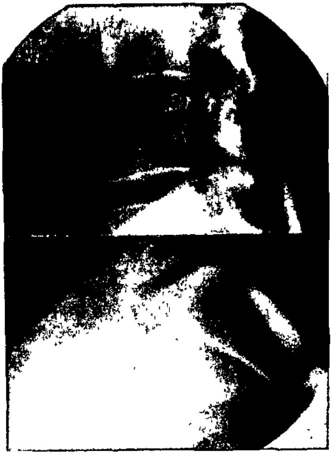


FIG. 18. Lateral roentgenogram 4 weeks after operation of the spinal fusion shown in Figure 16. The graft is in approximation with the remainder of the spine, the patient having been ambulatory since the 4th postoperative day.

result. Good fixation of the graft and firm contact with its bed are very important for early union. Figure 16 shows an "H" graft being locked in the undercut spinous process above and below as a spinal fusion. When bone chips are used, they should be packed to form as near a homogenous mass as possible. Cancellous cultured calf bone becomes very soft after storage, and it should not be used where there is stress on the graft.

CLINICAL STUDIES

Early union of this type of bone graft has been the rule. Figures 17 and 18 are roentgenograms 4 weeks after operation of the graft shown in Figure 16. This patient was ambulatory on the 4th day after operation. These roentgenograms show the graft to be in good position, with evidence of early union at the ends of the graft. This case progressed to solid fusion in less than 4 months, as shown in Figures 19, 20, 21 and 22.

Cultured calf bone encourages union in old fractures rather than acts as organic splints. Figure 23, *left*, is a nonunion of both bones of the forearm of 1 year's duration in a 65-year-old female with osteoporosis. Double onlay grafts of cultured calf bone were used, and good union resulted in 8 weeks, as shown in Figure 23, *right*. There also was good function of the arm and the hand in 8 weeks. In a comparable case of nonunion of both bones of the forearm in a strong male, 22 years of age, frozen human bank bone was used as double onlay grafts. Thirty four weeks after operation, there



FIG. 19. Roentgenograms 4 months after operation of spinal fusion shown in Figure 16 *(Left)* Right lateral bending. *(Right)* Left lateral bending.

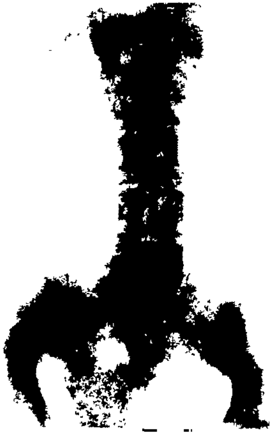


FIG. 20. Superimposed roentgenograms of right and left lateral bending demonstrating fusion of L 4 and 5 to the 1st sacral segment. This is the examination 4 months after operation of the fusion shown in Figure 16, in which a cultured calf bone graft was used.

still was nonunion, and the grafts had begun to be absorbed (Fig. 24).

To date, cultured calf bone has been used in the following cases:

Case 1. A 38-year-old female. Diagnosis: Spondylolisthesis. Lumbar fusion with "H" graft of cultured calf bone, L-3 to S-1. Clear serous drainage from the incision began 2 weeks postoperatively and healed spontaneously. Patient was ambulatory with lumbar belt on 4th day, and returned to work as a housemaid after 4 months.

Case 2. A 33-year-old male. Diagnosis: Herniated disk. Laminectomy and fusion with "H" graft of cultured calf bone L-3 to S-1. Was am-



FIG. 21. Roentgenograms 4 months after operation of spinal fusion shown in Figure 16. *(Left)* Lateral view with the spine in hyperextension. Note the increase in the thickness of this graft as compared with it 3 months previously in Figure 18. *(Right)* Flexion roentgenogram.



FIG. 22. Superimposed roentgenograms of extension and flexion of the spine, demonstrating fusion of L 4 and 5 to the 1st sacral segment. This is the examination 4 months after operation of the spinal fusion shown in Figure 16, in which a cultured calf bone graft was used.

bulatory on 3rd postoperative day with a lumbar belt. Healed without complications, and returned to work in laboratory after 3 months. Roentgen evidence of fusion at 3 months.

Case 3. A 35-year-old male. Diagnosis: Herniated disk. Laminectomy and fusion with "H" graft of cultured calf bone L-3 to L-5. Ambulatory on 3rd postoperative day. Healed without complications, and returned to light work in rice field after 3 months. Roentgen evidence of fusion in 3 months



FIG 23. *(Left)* Nonunion of both bones of the forearm, 1-year's duration, in a 65-year-old female with osteoporosis. *(Right)* Double onlay grafts of cultured calf bone showing good union 8 weeks after operation.

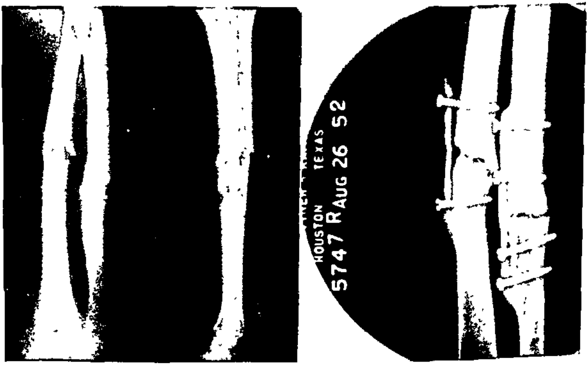


FIG 24 *(Left)* Nonunion of both bones of the forearm, 1-year's duration, in a 22-year-old male in excellent physical condition. *(Right)* Double onlay grafts of frozen human bone In 34 weeks there still is no union of the fracture, and the graft is undergoing absorption and fragmentation.

Case 4. A 65-year-old female. Diagnosis: Nonunion of middle one third of ulna and radius of 1 year's duration with osteoporosis. Double onlay grafts of cultured calf bone. Healed without complications. Good union and function after 8 weeks.

Case 5. A 68-year-old female. Double spondylolisthesis, L-3 and 4 on L-5. Advanced osteoporosis. Lumbar fusion with calf bone "H" graft 8 in. long, L-1 to L-5. Ambulatory with lumbar belt on 4th postoperative day. Clear serous drainage after 2 weeks, and lasted 2 weeks. Returned to tour as missionary in 3 months. Roentgen evidence of fusion in 4 months.

Case 6. A 31-year-old female. Diagnosis: Tuberculous spondylitis T-12 and L-1. Long "H" graft fusion of cultured calf bone from T-10 to L-2. Ambulatory on 3rd postoperative day with long Hoke canvas back brace. Healed without complications and returned to light housework in 1 month. Roentgen evidence of fusion in 4 months.

Case 7. A 47-year-old female. Herniated lumbar disk. Laminectomy and fusion with "H" graft of cultured calf bone L-4 to S-1. Patient ambulatory with lumbar belt on 5th postoperative day. Healed without complications. Roentgen evidence of fusion in 3 months. Returned to work as schoolteacher in 3 months.

Case 8. A 42-year-old male. Spondylolisthesis L-4 on L-5. Spinal fusion "H" graft with cultured calf bone L-3 to S-1. Patient ambulatory on 4th postoperative day with lumbar belt. Healed without complications. Returned to laboratory work in 8 weeks. Roentgen evidence of fusion in 4 months.

Case 9. A 42-year-old female. Herniated lumbar disk. Laminectomy and fusion with "H" graft of cultured calf bone L-3 to L-5. Patient ambulatory on the 4th postoperative day with lumbar belt. Healed without complications. Light housework after 8 weeks. Roentgen evidence of fusion in 4 months.

Case 10. A 36-year-old male. Herniated disk. Laminectomy and fusion with "H" graft of cultured calf bone L-4 to S-1. Patient ambulatory on 3rd postoperative day. Healed without complications. Returned to work as a salesman in 8 weeks. Roentgen evidence of fusion in 14 weeks.

Case 11. A 47-year-old female. Poliomyelitis residual dropped foot. Triple arthrodesis and posterior bone block with cultured calf-bone graft to posterior surface of the tibia. Graft was separated from the tibia in 3 severe falls suffered by the patient. Graft remained in soft tissue with fibrous union to tibia after 1 year, and was removed later.

Case 12. A 44-year-old female. Herniated lumbar disk. Laminectomy and fusion with "H" graft of cultured calf bone L-4 to S-1. Patient ambulatory on 6th postoperative day. Healed without complications. Returned to work as secretary in 12 weeks. Roentgen evidence of fusion in 5 months.

Case 13. A 54-year-old female. Nonunion lower one third of humerus of 11 months' duration, osteoporosis and diabetes mellitus. Healed without complications. Union in 10 weeks.

Case 14. A 46-year-old male. Herniated lumbar disk. Laminectomy and fusion with "H" graft of cultured calf bone L-4 to S-1. Ambulatory on 4th postoperative day with lumbar belt. Healed without complications. Returned to work on printing press after 4 months. Roentgen evidence of fusion in 4 months.

Case 15. A 34-year-old male. Extradural lipoma hypertrophied ligamentum flavum. Laminectomy and fusion with "H" graft of cultured calf bone L-3 to L-5. Patient ambulatory on 3rd postoperative day with lumbar belt. Healed without complications. Returned to light work in refinery after 6 weeks. Roentgen evidence of fusion after 3 months.

Case 16. A 27-year-old female. Herniated lumbar disk 3. Laminectomy and fusion with "H" graft of cultured calf bone L-2 to L-4. Patient ambulatory on 4th postoperative day with a lumbar belt. Wound healed in 14 days without complications. Returned to light housework after 18 days. Roentgen evidence of fusion in 14 weeks.

Case 17. A 38-year-old male. Herniated lumbar disk L-4. Laminectomy at L-4 and fusion from L-3 to L-4 with "H" graft of cultured calf bone. Patient ambulatory on 3rd postoperative day. Sutures removed on 12th day. Wound healed by primary intention. Patient returned to job as oil-field supervisor wearing lumbar belt on 18th day. Roentgen evidence of fusion in 14 weeks.

Case 18. A 75-year-old male (retired). Herniated lumbar disk L-3 with arthritic degeneration and collapse of pedicles at L-3 and L-4. Laminectomy of L-3, fusion with "H" graft of cultured calf bone L-2 to L-5. Patient ambulatory on 3rd day with long lumbar belt. Sutures removed on 12th day. Wound healed by first intention. Roentgen evidence of fusion in 12 weeks.

Case 19. A 34-year-old male. Spondylolisthesis L-5 on S-1. Exploratory laminectomy L-5. Fusion with "H" graft of cultured calf bone L-4 to S-1. Patient ambulatory on 3rd day with lumbar belt. Wound healed by primary intention in 10 days. Returned to work as engineer supervisor in 21 days. Roentgen evidence of fusion in 12 weeks.

Case 20. A 39-year-old female. Disk degeneration L-5 and mechanically unstable spine. Exploratory laminectomy L-5, lumbar fusion with "H" graft of cultured calf bone L-4 to S-1. Patient ambulatory on 4th day with lumbar belt. Wound healed by primary intention on 14th day. Returned to light housework in 18 days. Roentgen evidence of fusion in 14 weeks.

Case 21. A 33-year-old female. Large herniated disk L-4. Laminectomy L-4, fusion L-3 to L-5 with "H" graft of cultured calf bone. Patient ambulatory on 4th day with lumbar belt. Wound healed without complications in 12 days. Patient returned to light housework in 18 days. Roentgen evidence of fusion in 14 weeks.

Case 22. A 41-year-old female. Degenerated disk L-5 with spondylitis. Laminectomy L-5, fusion with "H" graft of cultured calf bone L-4 to S-1. Patient ambulatory on 4th day with lumbar belt. Wound healed without complications in 14 days. Returned to light housework in 16 days. Roentgen evidence of fusion in 14 weeks.

DISCUSSION

"Surgical graft: To implant living tissue so as to form organic union" (Webster). This definition is adhered to in the transplantation of many soft tissues such as skin, cornea, etc. However, the very nature of bone itself often makes one lose sight of the fact that it, too, is a viable and complex cellular structure. Therefore for the best results in bone transplantation, the cellular elements and the structure should be kept in as nearly unaltered a state as possible.

When we store bone at a low metabolic temperature, bathed in its own body fluid, we are preserving it with the least possible damage to the tissue itself. The storage of bone in its own nutrient media is a decided advantage for bacterial control. Should there be any infection in the bone, this fluid serves as a media for its detection.

The author realizes that this series of 22 cases of cultured calf bone is not large enough to demonstrate all the complications that may arise from this procedure, yet it is significant that there were no postoperative infections and no nonunions. Still the most important finding is the phenomena that when bone is preserved in its own plasma at a low metabolic temperature, it may then be transferred from one species to another without heterogenous tissue reactions, and it induces earlier union than autogenous bone.

SUMMARY

Experimental Results

By preserving calf bone in bovine plasma at a low metabolic temperature it was found experimentally that:

1. The bone cells remain intact for long periods of time.
2. An unorganized elastic and collagenous fibrous proliferation occurs on the cut surface of the bone.
3. There is little alteration of the structure of the bone.
4. Freezing of bone destroys the bone cells and alters its structure.
5. There is no untoward reaction between human bone preserved in plasma and calf bone; and no untoward reaction between calf bone preserved in plasma and human bone.
6. There is marked reaction between frozen human bone and calf bone.
7. Heterogenous bone preserved in plasma unites more rapidly than frozen bone.

Clinical Results

In the 22 cases in which cultured calf bone was used, the following results were observed:

1. There was no reaction between cultured calf-bone grafts and human being's.
2. Nonunion did not occur in this series with the exception of 1 case of posterior bone bloc in which the graft was separated from its bed by trauma.
3. Clear serous drainage occurred in 2 cases in which unusually large grafts were used. These cases healed spontaneously, and the serous drainage did not interfere with the union of the graft.
4. There were no postoperative infections.

CONCLUSIONS

Experimental and clinical experiences on the use of cultured calf bone have been reported.

Cultured calf bone was found to be a satisfactory bone graft for the following reasons:

1. Cultured calf bone can be made available in unlimited quantities.
2. This type of bone is stored and transported easily.
3. There is freedom from latent syphilis and from a hepatitis virus transmission.
4. Sterility of the bone graft is more positive, there being no postoperative infections in this series.
5. Cultured calf bone encourages early union, and there is no heterogenous bone reaction, even when large quantities are used in human beings.

REFERENCES

1. Axhausen: Die histologischen und klinischen Gesetze der freien Osteoplastik auf Grund von Thierversuchen, Arch. klin Chir. 88:109, 1909.
2. Barth, A.: Histologische Untersuchungen uber Knochenimplantationen, Beitr. path. Anat. 17:65, 1895.
3. Baschkirzew, N J., and Petrow, N. N.: Beiträge zur freien Knochenuberpflanzung, Deutsche Ztschr. Chir. 113:490, 1912.
4. Dobrowolskaja, N. A.: On the regeneration of bone in its relation to the cultivation of bone tissue, Brit. J. Surg. 4:332, 1916.
5. Esnaurrizar, M. L.: Heterogenous bone grafts, J. Internat. Coll. Surgeons 3:151, 1940.
6. Guilleminet, M., Stagnara, P., and Perret, T. D.: Preparation and use of heterogenous bone grafts, J. Bone & Joint Surg. 35B:561, 1953.
7. Haas, S. L.: A study of the viability of bone after removal from the body, Arch. Surg. 7:213, 1923.
8. Judet, J., Judet, R., and Arviset, A.: Banque d'os et hétérogreffe, Presse méd. 57:1007, 1949.
9. Ollier, L.: Traité des résections, Paris, G. Masson, 1885.
10. ———: Surgical bone grafting with os purum, os novum and boiled bone, J. Bone & Joint Surg. 19:873, 1937.
11. Orell, S.: Interposition of os purum in osteosynthesis, Surg., Gynec. & Obst. 59:638, 1934.
12. Schmitt, A.: Ueber Osteoplastik in klinischer und experimenteller Beziehung, Arch. klin. Chir. 45:401, 1893.
13. Terhune, S. R., and Shannon, P. W.: Personal experience in the use of homogenous and beef bone implants; a supplementary report, J. Lousiana M. Soc. 105:485, 1953.
14. Tucker, E J.: The preservation of living bone in plasma, Surg, Gynec. & Obst. 96:939, 1953.
15. van Meekeren, Job J.: Heel—en geneeskonstige aanmerkingen, Amsterdam, C. Commelijn, 1668. Abstr. Morton, L. T.: Garrison and Morton's Medical Bibliography, ed. 2, p 504, New York, Argosy Book Store, 1954.

Studios Super le Uso de Culturate Osso de Vitello in Graffos Ossee Human

Summario in Interlingua

Il esseva constatate que preservar osso de vitello a 40° F in plasma bovin rende tal osso compatibile con humanos.

Le osso es obtenite ab juvene, san vitellos sub le plus stricte conditiones aseptic. Illo es placiate in un grande receptaculo sterile e coperite con un solution salin physiologic

continente 20 pro cento plasma bovin. Antibioticos—un million unitates de penicillina e duo grammas de streptomycina per litro—es addite al solution. Un portion del medio es retirate pro tests post duo septimanas Con illo, culturas es executate pro organismos aerobie e anaerobie. Quando omne culturas es negative, le osso es placiate in receptaculos individual, coperite con plasma fresc, e culturas in bouillon a thioglycollato es executate pro omne receptaculo como controlo final.

Sero pro intradermatic tests de sensibilitate debe esser obtenite con le osso. Leve o moderate reactiones de sensibilitate pote esser negligite, sed un test fortemente positive contro-indica le uso del osso in question.

Factores essential in le utilisation del graffo es stricte asepsis, bon sanitate del substructura, exacte approximation, e alte grado de immobilisation. In conformar le graffo on se servi de un tenalia-vite e un serra manual al tabula de operation. Le graffo es protegite contra le maxillas del tenalia-vite per pecias de gaza. Serras a motor resulta in necrosis de calor que pote causar non-union.

Iste typo de graffo ossee esseva usate in 22 casos, sin occurrentia de infectiones postoperative e sin un sol non-union. Le osso usate stimulava union in ancian fracturas, e un prompte union del graffo esseva le norma. Grande quantitates de culturate osso vitellin in fusiones spinal es ben tolerate per le organismo human.

18

Central Fractures of the Acetabulum*

ROBERT B. ELLIOTT, M.D., F.A.C.S.†

It is the author's purpose to review briefly the discouraging, and yet challenging, problem of central fractures of the acetabulum and to present a method of internal fixation that he believes to be new, both in application and in principle, in the treatment of this fracture. It is hoped that it will add to our armamentarium in the treatment of this problem and stimulate further interest in and study of this type of fracture.

Central fractures of the acetabulum really are fractures of the pelvis, but the symptoms and the disability are those of the hip joint itself. The subject is classified in various textbooks as pelvic fractures or hip fractures and dislocations. In all the texts studied, except Key and Conwell[6] (who devote about 6 pages to this subject), this fracture is dismissed in a paragraph or two, or a page or two at the most. All texts studied had in common an outline of conservative treatment with traction, but, except for citing an individual case experience now and then, none had any proposed or outlined operative treatment in detail. Review of the literature revealed more interest in the subject, but most studies published were of findings, complications and end-results, and only a few mentioned specific operative treatment

Usually, the mechanism of injury is force applied to the greater trochanteric region, the head of the femur being driven centrally against the floor of the acetabulum and fracturing the acetabulum. It is seen usually in young or middle-aged adults, since in the older age groups the neck of the femur fractures first as a rule. This chapter will be confined to central fractures of the acetabulum and will not include fractures of the posterior and the posterosuperior rims. Fractures of the floor of the acetabulum vary from mild, with fracture lines present but little or no displacement, to severe, with shattering of the acetabular floor, central intrapelvic displacement of the fragments and, in extreme cases, the intrapelvic subluxation and dislocation of the femoral head.

In this type of injury, both the head of the femur and the acetabulum itself sustain damage. Roentgenographically and clinically, the head of the femur does not usually show damage unless a piece has been fractured off the head, which occurs rarely. However, damage to the femoral head may have been sustained, even though not manifest, through damage to the blood vessels of the capsule and the femoral neck itself, particularly the posterior retinacula; through thrombosis of the intramedullary vessels in the head and the neck and intra-osseous vessels of the haversian system; or perhaps through an intracellular molecular change from impact to the femoral head, as suggested by Steward and Milford.[8] The usual damage seen is sustained by the acetabulum with fracturing disrupting the acetabular contours, frequently in the area of acetabular weight-bearing, and also central displacement of the inner table

* Read at the annual meeting of The American Fracture Association held at Houston, Texas, October 11-14, 1954

† Houston, Texas.

TABLE 1.
REPORTED 204 CASES OF FRACTURE-DISLOCATION OF THE HIP WITH 116 CASES ANALYZED. OF THESE, 20 (17.2%) WERE CENTRAL FRACTURE-DISLOCATIONS IN TYPE*

	15 Cases Treated Conservatively	5 Cases Treated Surgically
Excellent	0	0
Good	1	0
Fair	4	0
Poor	10	5
Avascular necrosis	8 (40%)	
Traumatic arthritis	11 (55%)	

* After Thompson and Epstein.[9]

allowing the femoral head to subluxate or even dislocate centrally. Due to the high incidence of avascular necrosis and traumatic arthritis reported as complications of this fracture, it is extremely important to restore anatomically the articular surfaces of the acetabulum. It is felt that the method reported herein represents another type of solution of this problem or approach to it in certain of these cases.

The incidence of avascular necrosis and traumatic arthritis following this type of fracture is very high and contributes largely to the poor results obtained. In 1951, Thompson and Epstein (Table 1) reported a series of 116 cases of hip dislocations, 20 of them being central fractures of the acetabulum with subluxation or dislocation of the femoral head Of these 20 cases, 15 were treated conservatively and 5 were treated surgically, the method of surgical repair not being mentioned. There were *no* excellent results in their series; only 1 good result—a case treated conservatively—and 19 fair or poor results. Of these, avascular necrosis developed in 8 cases, and traumatic arthritis developed in 11 cases They concluded that:

> There is no suggestion that open procedures will save the joint from the changes inherent in avascular necrosis of the femoral head. However, in certain instances there is reason to believe that, if incongruity of the articular surfaces can be avoided by restoring the position of the fragments necessary for stability or by removal of loose fragments, it will be possible to reduce the incidence of traumatic arthritis. There are a certain number of cases in which this assumption has been borne out.[9]

TABLE 2. REPORTED 101 CASES OF FRACTURE-DISLOCATION OF THE HIP*

	Averaged 26% in all cases, including all types
Incidence of traumatic arthritis	15% in cases of dislocation of the hip without fracture
	25% in cases of dislocation with associated fracture of the acetabular rim
	60% in cases of dislocation with the head of the femur fractured
	100% in cases of dislocation where the acetabular floor was fractured

* After Armstrong, 1948.[1]

In 1948, Armstrong (Table 2) reported a series of 101 cases of hip dislocations, and found the over-all incidence of traumatic arthritis in all types of cases to be 26 per cent. However, in breaking this down into various types, he found the incidence to be 100 per cent in cases of dislocation when the acetabular floor was fractured.[1]

Steward and Milford (Table 3) followed 128 cases, of which 28 (14%) were central fracture-dislocation types. Of these 28, 22 were analyzed: 18 were treated conservatively; 4 were treated by surgery, the method not mentioned. In their 18 conservative cases, they had 50 per cent good or better results and 50 per cent poor or fair results, with avascular necrosis developing in 3 cases (16.6%) and traumatic arthritis developing in 10 cases (55.5%). In the 4 surgical cases, there were no excellent results, 1 good result, 1 fair result and 2 poor results, with avascular necrosis developing in 2 cases (50%) and traumatic arthritis developing in 3 cases (75%).[8]

TABLE 3. STUDY OF 128 CASES OF FRACTURE-DISLOCATION OF THE HIP, 28 (14%) BEING CENTRAL FRACTURE-DISLOCATION OF THE ACETABULUM IN TYPE*

6 Cases Lost	18 Cases Treated Conservatively	4 Cases Treated Surgically
Excellent	2	0
Good	7	1
Fair	1	1
Poor	8	2
Avascular necrosis	3 (16.6%)	2 (50%)
Traumatic arthritis	10 (55.5%)	3 (75%)

* After Steward and Milford.[8]

In a series of 3 papers published in 1947 and 1948, Urist (Table 4) dealt with this subject in 2 of the papers. In his 2nd paper, he discussed a series of 8 cases of central fractures without dislocation of the femoral head and 3 cases of severely comminuted bursting fractures of the acetabulum that disorganized the entire acetabulum.

Of the 8 cases, 4 showed no displacement at all, 3 showed minimal displacement and 1 showed marked displacement of the inner table and intrapelvic protrusion of the femoral head, treated by traction methods. This last case was not available for follow-up, and he makes no mention of traumatic arthritis in the other 7 cases, except to state that they "showed a good functional result."

The 3 bursting cases all were poor results within 2 years and required arthroplasty or arthrodesis.[11]

In his 3rd paper, he described 27 cases, 8 of which were central fractures of the acetabulum with generally poor results noted from conservative treatment. He stated:

> In both groups of cases, the integrity of the lunate acetabular cartilage and the rim of the acetabulum is the critical factor in the end-results. In matched cases of fractures, treated conservatively and by open operation, good function and little or no disability were shown when the joint surfaces were restored as perfectly as possible, but this could be accomplished only by open reduction.[10]

TABLE 4. STUDY OF 11 CASES OF CENTRAL FRACTURE-DISLOCATION OF THE HIP (2ND OF 3 PAPERS)*

8 cases of central fracture of the acetabulum *without dislocation*, treated conservatively by traction	4 cases—no displacement 3 cases—minimal displacement 1 case—marked displacement
3 cases of acetabular fractures of the severe bursting type	All 3 cases had very poor results within 2 years time

In the 3rd paper: 27 cases of fracture-dislocation, 8 being of the central fracture type. All were treated conservatively; generally poor results noted.

* After Urist[10, 12] (series of 3 papers)

Thus, quoting Campbell's *Operative Orthopaedics:*

> It is essential that the normal anatomy of the hip be restored as nearly as possible. Even the slightest subluxation of the joint or incongruity of the weight-bearing surface of the acetabulum is not compatible with good function. Further, any incongruity in the acetabular wall or in the head of the femur is certain to produce a degree of traumatic arthritis, . . . and any displacement of the head of the femur is soon followed by degenerative changes in the joint.[2]

Writing in 1954, H. Herman Young stated *similarly:*

> Although irreparable damage may have taken place in the femoral head at the time of injury, the disruption of the acetabulum will most certainly lead to degenerative changes unless early and adequate reconstructive measures are instituted. Incomplete reconstruction of the acetabulum leads again to an unstable hip joint with a slipping and rubbing of the femoral head. Even an undamaged femoral head eventually will degenerate with resultant pain and limited action.[16]

From the above series of cases presented in the literature and conclusions drawn, it is apparent that central fractures of the acetabulum have a rather poor prognosis on

past methods of treatment. Avascular necrosis of the femoral head is a large part of the final disability, but it is exceeded in all series by those cases presenting traumatic arthritis, which becomes, therefore, the larger single cause of the poor results. Anatomic restoration of the acetabular fragments should decrease this incidence of traumatic arthritis. Several authors concur in this and point it out. Yet, in present-day treatment of this condition, and even in our present textbooks, little or no mention is made of surgical restoration of the anatomy of the acetabulum. The author believes that the restoration of the acetabular joint surfaces is just as important as the restoration of the ankle joint, the knee joint or elbow joint surfaces, and that the acetabulum has been ignored and has not received similar consideration and emphasis toward obtaining an anatomic restoration. A large series of cases is needed in which restoration of the acetabulum is performed and later analyzed. It is hoped that this report is such a start.

The classic treatment from a conservative standpoint has been traction in the longitudinal line of the leg with lateral traction on the hip, accomplished with a sling or a band round the thigh, or with skeletal traction from the greater trochanter, with a Kirschner wire or a Steinmann pin through the trochanter, or by means of a screw hook or eye inserted into the trochanter.[2,6,7,13] The old Whitman method of an abduction spica, using the greater trochanter as a fulcrum against the rim of the acetabulum, has largely been discontinued.[15] Digital reduction of acetabular fragments with a finger in the rectum has been condemned by several authors.[6] Closed anesthetic reduction is mentioned by Key and Conwell, both with and without plaster spica following, and with and without traction on the involved leg.[6] The Jahss turnbuckle-cast-leverage method also has been tried in some of these cases.[5,7]

All these methods are directly dependent on the capsular ligament attachments for their reduction of the fragments, and it is the capsule attached to the intertrochanteric line of the femur and the rim of the acetabulum that is pulled really. The femoral head will reduce frequently, but the inner table of the acetabulum rarely does, since it is not attached to the capsule and is uninfluenced by femoral traction. This failure to reduce the acetabular fracture itself usually has been ignored by most surgeons, who have preferred to treat the fracture conservatively and accept the supposedly inevitable bad result.

A few surgeons have performed open reduction on these fractures and reported on their manner of handling the fracture. Watson-Jones[13] quotes a personal communication from W. S. Diggle,[3] in which he approached the fracture from a mid-line suprapubic incision to expose the inner wall of the acetabulum, pressed the pubic fragment back in place, and fixed it with 1 screw into the ilium. Engel repaired the roof of the acetabulum through a Smith-Petersen anterior iliofemoral approach, tapping the fragments back in place with a bone set and a wooden mallet.[4] Levine presented a case with a similar operative approach, in which he replaced the acetabular fragments digitally and with a bone set; he held them with a bone plate of steel bent to fit the inner contours of the pelvis and fixed with 4 screws to the iliac bone, with the end of the plate spanning the acetabular floor and holding the fragments reduced.[7]

The most recent surgical treatment that has come to the attention of the author has been the cases presented by Anders Westerborn, of Sweden,[14] with severe bursting fractures of the acetabulum, in 6 cases of which he has performed primary Vitallium-cup arthroplasty, the cup being of such size that it did not recede into the acetabulum and thereby restored a smooth metallic acetabulum of normal size, shape and depth. His reported results in 4 cases appear to bear further investigation and look promising.

Additional review of the literature failed to disclose an approach to the problem and fixation of the fracture as undertaken by the author.

The most frequent type of fracture of the acetabulum seen presents the entire floor of the acetabulum swung inward (Figs. 1 & 2, *top*). The main fracture line runs into the upper part of the acetabulum, separating the ilium above from the pectineal portion of the pubis below, and it is the pectineal portion of the bone that is displaced inward from the ilium. This fracture line, therefore, usually involves the superior weight-bearing portion of the acetabulum. With exposure through a Smith-Petersen anterior iliofemoral approach, with detachment of the sartorius, the tensor fascia lata and the rectus femoris

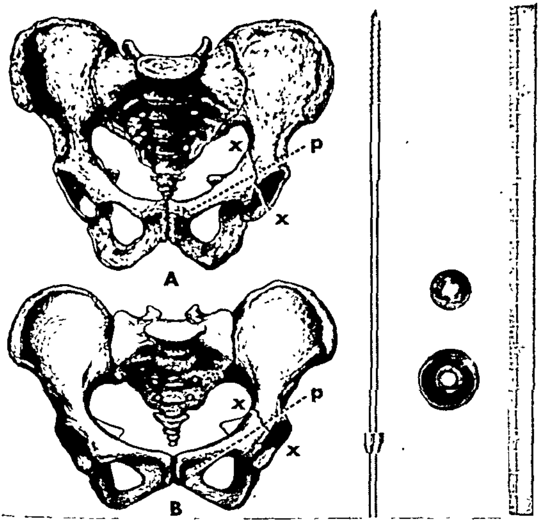


FIG. 1. This shows the gross anatomy of the pelvis and the acetabulum in the male and the female pelves *The line X----X shows the most common* type of acetabular fracture, with the main fracture line running into the acetabulum below through the thick acetabular roof, where the ilium joins the pectineal portion of the pubis. The fracture line continues posteriorly across the roof of the acetabulum and then proceeds distally, usually involving the posterior portion of the acetabulum. The line P - - - - - shows the placement of the Hagie pin which is used for fixation, it is inserted through the thick acetabular roof from the lateral aspect of the pelvis, across the fracture site, thence down the intramedullary canal of the horizontal ramus of the pubis. To the right is shown a Hagie pin, 6 in. long, with a ⅛ in. diameter threaded shank and a 5/32 in diameter threaded screw, made of SMO stainless steel and showing the nut reversed on the pin for greater surface bearing when tightened. This is a small lag screw in principle; it has strong grasping power and drills its own hole. The washers shown to the right may or may not be used, as indicated by the case. (Schaeffer: Morris' Human Anatomy, ed. 9, Blakiston-McGraw)



Fig. 2. *(Top)* Shows the fracture of the left acetabulum in the usual location, where the pectineal pubis joins the iliac bone. Only slight displacement and only slight central subluxation of the head of the femur are present. *(Bottom)* Shows the end-result after treatment with conventional longitudinal and lateral traction on the left leg. The acetabular contours are smooth, including the superior weight-bearing portion. Full range of hip motion is present. The hypertrophic changes seen in the hip joint preceded the injury, and there is no indication at this time of a superimposed traumatic arthritis.

origins, the superior and the anterior portions of the acetabulum come into view. With subperiosteal reflection of the iliacus medially from the inner side of the iliac bone and retraction medially of the conjoined iliopsoas tendon where it passes in its groove between the anterior inferior spine of the pelvis and the pectineal eminence of the pubis, the fracture site is exposed readily. The thick muscular fibers of the iliopsoas here give the femoral vessels and nerve adequate protection when retracted. The hip joint capsule and the head and the neck of the femur are not disturbed. If further exposure of the fracture site is desired, the iliacus muscle can be reflected further from the inner side of the pelvis, and adequate visualization is obtained. The fracture line can be opened more widely, and the joint thereby is exposed and debris is removed. The head of the femur can be inspected through this opening by movement and rotation of the femur. An anatomic reduction can be obtained by pushing the pectineal pubis fragment into place against the iliac bone and holding it there while fixation is achieved. The head of the femur usually reduces spontaneously with this maneuver or with slight longitudinal traction on the leg.

It was the author's opinion that intramedullary fixation of this fracture would be excellent if possible, and such was carried out using Hagie pins (Fig. 1), with which he had had previous experience in pinning subcapital fractures of the neck of the femur. This pin is a small lag screw in principle, 6 in. long, with a threaded shank ⅛ in. in diameter and a threaded screw $\frac{5}{32}$ in. in diameter. Tightening the nut on the shank causes firm strong contact compression of the fragments. The insertion is with a drill, with the pin started on the lateral pelvic wall above the superior rim of the acetabulum and directed medially through the thick portion of the acetabular roof until it emerges in the fracture end of the proximal fragment (ilium), then crosses the fracture line and penetrates medially down the intramedullary portion of the horizontal ramus of the pubis. The lag nut on the proximal end of the pin then can be tightened, either with or without washers, and an excellent stable fixation is achieved. Closure is in layers, with repositioning of the detached muscles in a routine fashion.

Examination of 8 skeletons revealed the roof of the acetabulum to be quite thick and capable of placement of this type of pin

(Figs. 1 & 8). The thickness varied from not less than ¼ in. to over ½ in. in all cases, with the thickness being closer to ½ in. in most of the skeletons. Examination of 2 cadavers confirmed the operative approach and the line of placement of the pins. Care must be taken to start the pin insertion above the superior rim of the acetabulum, so as to allow the pin to pass through the thick roof of the acetabulum above the uppermost portion of the joint surface and to avoid entering the superior portion of the acetabulum proper with the pin. The operation has since been carried out on 2 patients: there were no technical difficulties in the 2nd case; the only difficulties in the 1st case were from callus and scar formation already present, which prevented anatomic restoration.

CASE HISTORIES

Case 1. A 45-year-old Mexican fell, landing on his left hip and pelvis. Figure 2, *top*, shows the fracture of the acetabulum that was present. This case shows little or slight displacement and is typical of the cases that usually show an excellent end-result with only bed rest or traction treatment in the conventional manner.

Figure 2, *bottom*, shows the final end-result with complete healing present. The patient has a full range of motion in all directions, no pain or disability, no limp, and no complications such as avascular necrosis or traumatic arthritis. He has returned to his old job.

This case was lost to follow-up.

A second case similar to this has been seen and treated conservatively, with a similar excellent result. These fractures do *not* require surgery, and it should *not* be performed.

Case 2. A 46-year-old white female was injured in an automobile collision. She was riding sideways in the back seat and struck the back of the front seat with her left hip and pelvis. Figure 3, illustrates the fracture of the acetabulum sustained, with slight separation of the left sacro-iliac joint and the iliac fragment swung out laterally, instead of medial displacement of the pubic fragment, as is usual. Conservative treatment with longitudinal leg traction and a pelvic sling did not reduce the fracture. Pelvis binder round the iliac crests did not help.



FIG. 3. Shows the fracture of the left acetabulum sustained by a 46-year-old white female in an automobile collision. The superior articular surface portion of the acetabulum is involved. There is slight separation of the left sacro-iliac joint, and the iliac fragment has displaced laterally, instead of the central table of the acetabulum having been displaced medially, as is usually the case. However, the net result is the same, with a deepened acetabulum and disruption and roughening of the articular surface of the superior acetabulum. Initial treatment was with longitudinal and lateral left leg traction and with a pelvic sling, and later a pelvic binder, but no appreciable reduction was obtained.

On the 8th day after injury, open reduction was performed, and anatomic restoration was achieved with considerable difficulty. Fixation was by a Webb tibial bolt with a washer and a nut on each end, plus 1 stainless steel screw across the fracture line. This is shown in Figure 4, *left*.

The patient did not carry out postoperative orders and walked bearing full weight on the hip. Subsequently there were poor callus formation and absorption at the fracture site, as shown in Figure 4, *center*. She was put back on crutches without weight-bearing, and subsequently went on to full healing, as shown in Figure 4, *right*. Note, however, the irregularity of the acetabular joint surface and the beginning of a traumatic arthritis of this hip joint, which eventually will render this a poor functional end-result.

Because of the technical operative difficulties in the fixation of this fracture and in trying to fasten a washer and a nut on the end of a pin



FIG. 4. Surgery was performed on the 8th day after injury after failure of conservative traction and molding. *(Left)* Shows the reduction obtained at time of surgery and the fixation used in this case, which consisted of a Webb tibial bolt with a washer and a nut on each end, supplemented with a long bone screw. The restoration is almost anatomic. *(Center)* Shows the result 10 months later with poor callus formation and absorption at the fracture site. It is sometimes felt that synovial fluid contributes to this absorption and delayed healing, but the author believes that in this instance at least the picture is due to lack of co-operation by the patient, who bore full weight and resumed activities too early, with a shearing motion in the fracture line with each step. *(Right)* Shows the result 1 year later after the patient had been placed back on crutches with healing finally obtained, but with the area of absorption still present in the superior articular portion of the acetabulum Eventually this rough area will render this a poor functional result; some symptoms of traumatic arthritis are already appearing.

deep in the true pelvis, an easier manner of fixation was sought, with the evolution of the method presented at this time.

At present, 18 months afterward, this patient certainly is not any worse. Her hip aches with excessive weight-bearing and walking. She states that it aches when the weather is about to change. She is rather obese, and we are trying to obtain weight reduction, with only fair co-operation by the patient. Her motion range remains very good She is satisfied with the hip, does all her housework, takes care of her 5 children, and does everything she wants to do without limitation

Case 3. Figure 5, *left*, shows the original fracture sustained by a 28-year-old white male in an automobile collision, with the exact mechanism of injury unknown to the patient. It was felt that this was a case that could well be treated by conventional traction therapy, and such was applied. However, when the author saw this patient approximately 3 weeks after injury, the roentgenograms (Fig 5, *right*) showed that the fracture had come apart and displaced, and that the femoral head had subluxated slightly into the pelvis. Some flattening of the femoral head can be noticed on this view, indicating that there may have been some damage to the femoral head itself at the time of the injury. Additional conservative treatment methods failed to achieve any improvement in reduction.

Open reduction was performed on the 24th day after injury through an anterior iliofemoral approach. The fracture was reduced closely in the superior weight-bearing portion of the acetabulum, but it could not be restored anatomically because of the callus and scar-tissue formation present. Fixation was by means of 2 Hagie pins and washers, according to the method proposed.

This case was lost to follow-up.

Figure 6 shows the reduction obtained, the position of the pins, and the hip in anteroposterior and frog-leg lateral projections at the time of the last roentgenograms.

The patient is working full time at his old job as a laborer, and complains only that the hip tires easily, with some aching and soreness present toward the end of the day. Full flexion and extension are present, but very markedly limited internal and external rotation, and slight limitation is noted in abduction. The last roentgenograms show further degenerative arthritis of the head of the femur, which is felt to be due partly to the original trauma and also to the inability to restore anatomically the acetabular articular surfaces. It is felt that the hip will worsen gradually over the years.

Case 4. A 25-year-old white female was injured when the automobile in which she was riding missed a dead-end turn and went off the road into an embankment. Exact mechanism of injury was unknown to her. Figure 7 shows the roentgenograms of the original injury sustained by the right hip and acetabulum. There are a vertical splitting fracture of the acetabulum, a fracture of the posterior lip of the acetabulum and a posterior dislocation of the right hip. This patient also had a mild scoliosis of the spine and about 1 in. shortening of this right leg from old mild poliomyelitis involvement, with good muscular recovery but with slight shortening residual. Added complications of this injury were the presence of a brain concussion, multiple lacerations and contusions, and the fact that she was 2½ months pregnant at the time of the accident.

Immediate closed reduction of the hip dislocation was performed, and the leg was placed in traction. This is shown in Figure 8, *left*. Any further procedures at this time were contraindicated because of the patient's condition and the possibility of abortion of the fetus. Note in the



FIG. 5. *(Left)* Shows the original central fracture of the acetabulum sustained by a 28-year-old white male in an automobile collision. Initial treatment was by conventional longitudinal and lateral traction. *(Right)* Shows the appearance 3 weeks later at the time of consultation, the fracture having come apart and separated further. A second vertical fracture line now shows in the superior acetabulum surface. Also, some flattening of the femoral head can now be seen, indicating direct damage to it in addition to the damage to the acetabulum The prognosis of this hip joint was felt to be poor.



FIG. 6. Surgery was performed on the 24th day after injury with open reduction and pinning of the acetabular fragments with 2 Hagie pins. Good reduction was obtained in the superior weight-bearing portion of the acetabulum but failed otherwise because of the callus and the scar tissue already present at this late date. *(Left)* Shows the present result in anteroposterior projection *(Right)* Frog-leg lateral projection. Note the advancement of traumatic arthritis at this date, not only of the acetabulum, but also of the femoral head. The patient now has limitation of rotation but almost normal flexion and extension, and works full time at his old job as a laborer. Although his hip is not "normal," he is satisfied with it and meantime does not require the cup arthroplasty or fusion which eventually will be necessary.

roentgenogram the further separation of the acetabular fracture fragments even while in conventional traction.

The patient's condition improved, and the fetus showed no signs of being lost or endangered; therefore, open reduction and internal fixation were performed, on the 16th day after injury, through an anterior iliofemoral approach, as described, with fixation with 2 Hagie pins. Figure 8, *right*, shows the anatomic reduction obtained and the position of the Hagie pins holding the fracture. The hip remained stable in Buck's traction and showed no tendency to dislocate again. The posterior rim fracture fell into position and showed no tendency to slip or displace, and subsequently healed anatomically.

Weight-bearing on the hip was permitted about 4½ months after injury. The patient de-

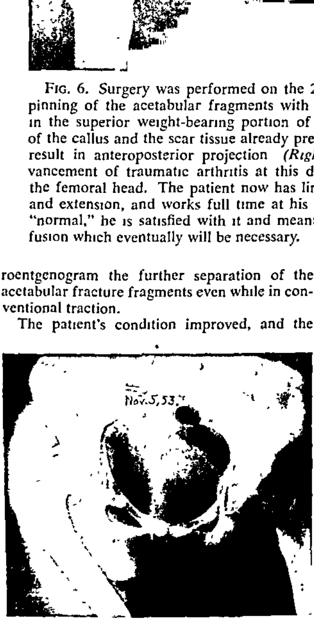


FIG. 7. Shows the original fracture-dislocation of the right hip and fracture of the acetabulum sustained by a 25-year-old white female in an automobile accident, in addition to other associated injuries and a first trimester pregnancy.

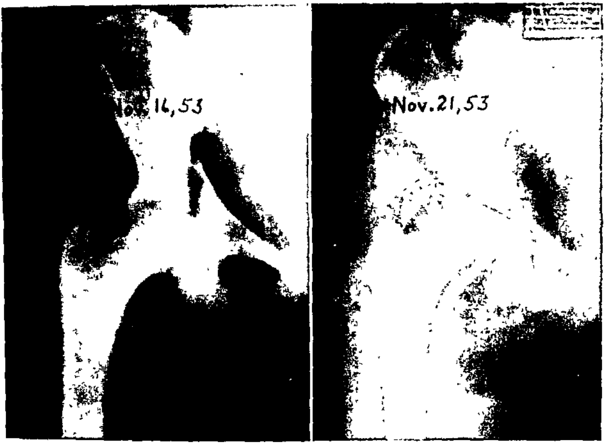


FIG. 8. *(Left)* Shows the right hip after closed manipulative reduction of the hip dislocation and traction. The condition now is similar to that seen in central fracture dislocation of the acetabulum. Note that with reduction of the hip dislocation and the pressure of the femoral head, the inner table of the acetabulum is pushed inward with further separation of the fracture fragments. *(Right)* Shows the postoperative appearance after open reduction and pinning with 2 Hagie pins. The restoration is anatomic. Note the position and the placement of the Hagie pins, running from the lateral aspect of the pelvis above the acetabulum through the thick acetabular roof, across the fracture line, and down into the intramedullary portion of the horizontal ramus of the pubis.

livered a healthy full term baby girl uneventfully about 7 months after injury. The last roentgenograms (Fig. 9) show her present status with the hip in anteroposterior and frog-leg lateral projections. She walks well in a fashion normal to her, with the same degree of limp that she had prior to the injury, and she cannot tell any difference in the hip now compared with what it was before injury. There is full flexion, full extension, full abduction and adduction, and normal internal and external rotation of the right hip Only occasionally does she have a dull, low grade ache in the hip, "when she stops to think about it," and no disability. She is doing all her household duties now, she has 3 children. The roentgenograms show solid anatomic healing and restoration of the acetabulum with no evidence of avascular necrosis or of traumatic arthritis as yet.

This patient is still rated as excellent in every respect. She was rechecked last in February, 1956, by roentgenograms. The hip is actually somewhat improved compared with what it was in September, 1954. Full range of all motions, no limitation. Minimal complaints: "It tires a little easier than the other hip"; "sometimes it aches a little when the weather is changing—not much, though"; "I am completely satisfied with my hip." Roentgenograms show no changes compared with those made in September, 1954 (Fig. 8), with no arthritis, no necrosis showing.

This case presented the most severe injury to the hip and the acetabulum of all the cases, and yet apparently the best restoration with surgery and the best result to date have been obtained.

To summarize: The treatment of central fracture of the acetabulum *without* displace-

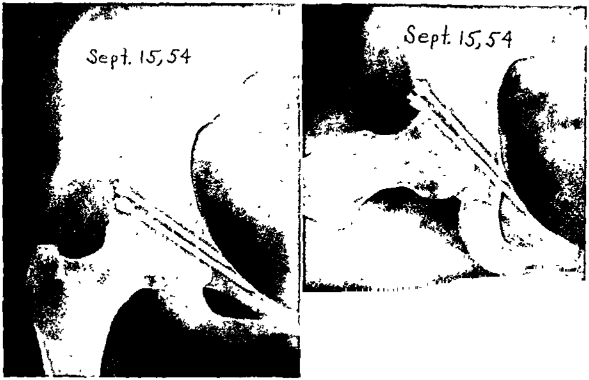


FIG. 9. These views show final healing status at this time. Note the anatomic restoration of the acetabular contours and the absence of traumatic arthritis. Although the Hagie pins appear to pass through the femoral head in the anteroposterior view *(left)*, they do not; they actually lie in the thick acetabular roof of the superior portion of the acetabulum, thereby being superimposed on the femoral head shadow. Proof of this is shown *(right)* by a frog-leg lateral projection of the hip with the femur flexed 90°. A complete range of motion is present in all directions at this time, and the present classification is excellent, although additional follow-up is indicated. The salvage of a badly shattered and disrupted hip with a poor prognosis appears to have been successful.

ment is handled best by the conventional method of longitudinal and lateral traction, and a good result usually can be anticipated. However, if the acetabular fragments *are displaced*, closed reduction and subsequent treatment in traction in extension will hardly ever give satisfactory results. It is the author's belief that in view of the poor prognosis with conservative treatment in such cases, they should be given the benefit of open reduction and internal fixation with anatomic restoration of the acetabulum and the femoral head Such restoration of the articular surfaces long has been emphasized in other joints; they are equally important in the hip joint.

Open reduction surgical treatment of this type of fracture is barely mentioned in current orthopaedic texts, and it is not detailed or outlined in any. A search of the literature has failed to show an approach to this problem fracture with intramedullary internal pin fixation as presented here by the author, and he believes that it is a new concept. It is certainly applicable to many of these fractures and, it is hoped, will improve the final end-results in many of them.

The time of operation after injury is extremely important, and surgical restoration must be undertaken *early* if anatomic reduction is to be successful, since it is practically impossible to obtain it after the fracture hematoma has clotted and fibrous scar tissue and callus have begun to form.

The treatment of the more severe bursting fractures by primary Vitallium-cup arthro-

plasty also is interesting and will bear further investigation. It is felt that results with conservative management previously reported now should be supplemented by a study of open reduction-acetabular restoration results.

It is hoped that this preliminary report of internal intramedullary pinning of central fractures of the acetabulum will stimulate further investigation and perhaps show a more favorable prognosis in a most unfavorable type of fracture.

REFERENCES

1. Armstrong, J. R.: Traumatic dislocation of the hip joint. Review of 101 dislocations. J. Bone & Joint Surg. 30-B:430-445, 1948.
2. Campbell: Operative Orthopaedics, edited by J. S. Speed and Hugh Smith, ed. 2, vol. 1, St. Louis, Mosby, 1949.
3. Diggle, W. S.: Quoted by Watson-Jones.
4. Engel, G. C.: Central dislocation of the head of the femur, Surg. Clin. North America 20:1724-1725, 1940.
5. Jahss, S. A.: Quoted by Levine.
6. Key and Conwell: Fractures and Dislocations and Sprains, ed. 5, St. Louis, Mosby, 1951.
7. Levine, Max A.: A treatment of central fractures of the acetabulum, J. Bone & Joint Surg. 25:902-906, 1943.
8. Steward, Marcus, J., and Milford, Lee W.: Fracture-dislocation of the hip. An end-result study. J. Bone & Joint Surg. 36-A: 315-342, 1954.
9. Thompson, V. P., and Epstein, H. C.: Traumatic dislocation of the hip. A survey of 204 cases covering a period of 21 years. J. Bone & Joint Surg. 33-A:746-778, 1951.
10. Urist, M. R.: Fracture-dislocation of the hip joint The nature of the traumatic lesion, treatment, late complications, and end-results. J. Bone & Joint Surg. 30-A: 699-727, 1948.
11. ———: Fractures of the acetabulum. The nature of the traumatic lesions, treatment, and 2-year end-results. Ann. Surg. 127: 1150-1164, 1948.
12. ———: Injuries to the hip joint. Traumatic dislocations incurred chiefly in jeep accidents in World War II. Am. J. Surg. 74:586-597, 1947.
13. Watson-Jones, Sir Reginald: Fractures and Other Bone and Joint Injuries, ed. 4, Baltimore, Williams & Wilkins, 1952.
14. Westerborn, Anders: Central dislocation of the femoral head treated with mold arthroplasty, J. Bone & Joint Surg. 36-A:307-314, 1954.
15. Whitman, R.: The treatment of central luxation of the femur, Ann. Surg. 71:62-65, 1920.
16. Young, H. Herman: Lesions of the hip joint: primary acetabular pathologic changes and primary synovial changes, Proc. Staff Meet. Mayo Clin. 29:41-43, 1954.

Fractura Central del Acetabulo

Summario in Interlingua

Fracturas central del acetabulo es usualmente associate con prognoses pauco favorabile. Le mal resultatos es principalmente debite a arthritis traumatic del articulation coxal o a necrosis avascular del capite femoral. Methodos conservative de tractamento non succede in general a restaurar le acetabulo anatomicamente e a reducer le procentage de secundari arthritis traumatic tanto del acetabulo mesme como etiam indirectemente del capite femoral. Le reconstruction chirurgic del acetabulo es pauco mentionate in le manuales e le litteratura periodic, sed le presente autor es convincite que illo es un methodo que merita plus investigation e application practic.

Es presentate un methodo pro le reparo chirurgic del acetabulo. Le accesso antero-iliofemoral de Smith-Petersen es usate. Le fixation es complite per medio de clavos transossee. Le typo de clavo usate es illo de Hagie. Illo es inserite ab le aspecto lateral del ilio, a transverso le spisse tecto acetabular justo supra le articulation coxal proprie, postea a transverso le linea del fractura, e finalmente a basso in le canal intramedullari in le rammo horizontal del osso pubic.

Es presentate duo casos illustrative. Nos sublinea le importantia de un prompte execution del operation, ante le formation de texito cicatrisante o de callo.

Nos opina que iste methodo de reparo e de fixation promove le restauration anatomic e assi tende a reducer le frequentia de arthritis traumatic que es, statisticamente, le plus importante causa de disfavorabile resultatos final. Avantages additional es le minus extense hospitalisation e un plus prompte ambulation. Il pare que iste methodo offere un melior prognose pro iste typo de fractura problematic.

19

A Search for Nonallergic Polymerizing Plastic

MILTON C. COBEY, M.D., F.A.C.S.*

The author's search for an agent that would form a solid light material began some 15 years ago. He realized that this would have to be done through the act of cold polymerization for use in the human being. If there were heat of polymerization, this would burn the patient externally, or, if it were used internally, it would burn tissues. In 1935, the popularly known *wing dope,* a cellulose acetate compound used for the covering of wings of airplanes, was the first cold polymer with which the author had close contact. With this material there could be formed a sheet of plastic that was not caustic to the human being and did not produce much heat of polymerization. This plastic would take a mold and form the shape of the extremity to which it was applied. It was originally demonstrated and presented at the American Medical Association meeting in 1937, and again at the American Academy of Pediatricians in Washington, D. C., in 1940. The objective was to prepare a waterproof, or at least a water-resisting, type of splint for children. Its use was in anterior poliomyelitis in the rest and protection treatment of paralyzed muscles. Casts up to that time were unsuccessful, as they absorbed moisture and soon became soft and no longer usable. Other compounds, such as shellac and other types of paint, were unsuccessful because they did not cover the plaster with a good strong coat of material. Since then, nylon has been used quite successfully for this purpose in place of the cellulose acetate molds.

From 1942 to 1946 lucite was used to advantage. Its transparency and its lightness made it excellent material with which to work. The author first prepared a retractor through which light could be passed and was used for knee surgery. In making this plastic into the proper curves for use in the knee joint, it was found that lucite could be heated and fashioned to about any desired shape and allowed to cool, the result being a dependable splint. Since then, many lucite splints have been available on the commercial market. A plaster mold can be made of the desired extremity or body. This is filled with plaster to make a male mold, then a lucite splint or mold can be made from that. This was used most successfully, particularly in areas in which there was constantly a high humidity and casts not only went to pieces because of absorption of moisture but were most uncomfortable and caused irritation of the skin because of this moisture. The difficulty remained that the plastic shell still required the use of a male and a female mold. Subsequently, several other materials were developed by the Mellon Research Foundation. None of these could avoid the mold until the properties of the melamine resins were discovered.

---

*Professor of Orthopedic Surgery, Georgetown University Medical School.

Associate investigators: Dr. Robert C. Rush, Assistant Professor of Orthopedic Surgery, Georgetown University School of Medicine; Dr. Herbert D. Lane, Jr., Research Fellow in the Study of Arthritis, Georgetown University; Dr. Charles F. Geschickter, Professor of Pathology, Georgetown University School of Medicine.

A melamine resin is a cold-setting polymer. No heat of polymerization is produced. It was found that its tremendous gluing properties could glue together tiny particles of plaster of Paris into a hard waterproof mixture that was quite malleable when wet, and its setting time could be controlled. This was used with the catalytic agent ammonium chloride, the best catalytic agent found. This was a real step forward, because for the first time a waterproof plaster was obtained. It would not absorb moisture in any way. It was 4 times stronger than plaster of Paris. Also, now it was possible to apply to the patient for external fixation of fractures a waterproof type of cast that was largely nonirritant to the skin and no more irritant than plaster of Paris. This could be used in all climates. The rapid setting properties and initial strength, as well as later strength, of the new plastic made for a light strong cast that was most gratifying.

Thus, a satisfactory method of external fixation of fractures, through the use of developed plastics, led one to think of the possibilities of internal fixation of fractures by the plastics also. At the present time, formed plastics, mostly of acrylic resins, are possible as prostheses. While not as strong as Vitallium or SMO steel, they have been used successfully in hip-joint surgery and in shoulder-joint surgery in the form of replacement prostheses for the heads of these bones. Nylon and other plastics have been used and have met with varying success. The idea of a cold polymer for internal fixation originated when the cold polymerizing action of the melamine resins became known. However, the acrylics throw off a great deal of heat, and for this reason they are not usable inside the body. The heat of polymerization of the acrylic resins is sufficient to burn tissues. The melamine resins did not prove to be successful inside the body, largely because of the formaldehyde content. This was sufficient to cause a necrosis of tissue. But with the aid of many research laboratories in the plastic field, studies were begun to evolve a plastic that could be used in this project. It was necessary to study the toxicity of any new plastic that we made or could obtain. This was done with 220 to 300 Gm. rats. It was also necessary to have a plastic mass that would become hard through the act of polymerization inside the body. This mass then had to become softened after a period of 4 to 6 weeks in order for it to become absorbed through the body. During this absorptive, or hydroscopic, period, a new protein matrix would have to grow between the bone ends and be invaded by calcium salts in order to produce healing of the fracture in the case of internal fixation of fractures.

These 4 ideas were kept in mind in trying to develop a new type of plastic. The chart on page 208 lists the plastics that have been made and/or tried by us in this extensive study. For its size, the chart is as accurate as possible, but of course it requires explanation. The materials are outlined in the chart as they were used by us in progressing step by step to the final gel that we now have; while it is not altogether successful, it shows promise.

Elvanol was used first because it was a commercial product that was available and had been suggested by some work that had been done in Britain. Elvanol is used in film-making commercially; it is a polyvinyl alcohol and can be changed to the acetate emulsion, becoming polyvinyl acetate emulsion, 55 per cent of which is solid. This was found to be nontoxic, as high as 7 cc. to the rat. The material had cementlike properties, and would set immediately in the syringe. In its pure alcohol solution, Elvanol was found to be somewhat toxic, but the acetate would remain an unabsorbed gel at a fracture site for as long as 3 months. It would set much quicker at the fracture site if used with glycerin, which would act as a hydroscopic agent. However, it was never possible with this gel to hold the bone ends together as a complete internal splint.





FIGS. 1 to 4. The plastic Elvanol as shown *in situ* at the fracture site, the injection method used in this research work in rats. (Figs 3 & 4—Cobey, Milton C.: J. Internat. Coll. Surgeons 25:92)







FIG 5. This is a microscopic section showing the bone fragment with cellular response to fracture and plastic.



FIG. 6 Microscopic marked granulation response with foam-cell reaction (Cobey, Milton C J Internat Coll Surgeons 25:93)



FIG 7. Cellular muscle necrosis with granulation tissue as a toxic reaction to irritant type of plastic, such as sodium polyacrylate.



FIG. 8 Plastic material in juxtaposition with muscle tissue with peripheral cellular response, demonstrating cellular response without necrosis.

Because of the work of Dr. S. Kiser, and of Dr. Knute Jansen, of Copenhagen, sodium polyacrylate was investigated. This acrylic compound was found to be very toxic, and it lacked the property of cold setting as well. It had been used by these authors as a glue to fix the Judet type of plastic head to the neck of the femur. They allowed it to be heated to high temperatures, to become soft and then to give off heat as it set. Since it set very quickly, they felt that the small amount of necrosis did not do harm. This, of course, would not be usable at a fracture site in a human being if it were injected inside the body.

Figures 1 to 4 represent Elvacet, or the acetate of Elvanol *in situ*. Microscopically, the reaction is one of foreign bodies, but there is no evidence of any malignant change. Present are macophages, which may be the precursor cell to chondroblasts, chondroclasts, osteoblasts and osteoclasts. The various compounds were very toxic to rats, but it was necessary to try them out in an effort to determine whether or not that type of plastic could be used. It is noted that wherever calcium was used in the compound, roentgenograms could be taken of the fracture site and the injection site to ensure that the material was injected at the proper place. The calcium shows up very well on the roentgenogram. The single needle method of injection, doing it quickly when the material was still in its liquid state in a syringe, often was rather difficult and never possible to sterilize. So when the study continued to the use of dextran and glycerin with the polyvinyl methyl ether maleic anhydride copolymer, the two needle method of injection was adapted; that is, injecting the polymer and then the catalyst with the second needle. It was possible then to sterilize the compounds, and any slough that occurred would not be the result of infection. The most successful material, as one will see from the chart, turned out to be the seaweed, or Irish moss, solutions. These could be injected with the polymer in 1 syringe and a salt solution in the other syringe, as will be seen by the list of the most successful catalysts. The salt compounds supplied not only an available amount of calcium to the fracture site but the catalytic agent for cold polymerization rapidly at the fracture site in a nontoxic solution.

PARTIAL LIST OF CATALYSTS USED

The early test salts had the following composition:

Calcium carbonate, M.W. 100	10 0 Gm.
Magnesium oxide, M.W. 40	0.4 Gm.
Phosphoric acid, M.W. 98	10.0 Gm.
Citric acid, M.W. 192	10.0 Gm.

Water to make a paste, stirred and neutralized *with sodium hydroxide to pH around* 5.

Later the composition was changed to include strontium:

Calcium carbonate	10.0 Gm.
Magnesium oxide	0.4 Gm.
Strontium carbonate	15.0 Gm.
Phosphoric acid	10.0 Gm.
Citric acid	10.0 Gm.

This mixture was prepared as indicated above. Toxicity troubles indicated that a change was needed, and the following resulted:

Calcium carbonate	5.0 Gm.
Magnesium oxide	0.5 Gm.
Strontium carbonate	0.5 Gm.
Phosphoric acid	4.0 Gm.
Citric acid	4 0 Gm.

Slurried and pH adjusted as indicated above.

EXPLANATORY CHEMICAL NOTES

In all cases the slurries were mixed with protein-solution vehicle. It was noted that autoclaving depolymerized the gelatin and gelatin-collagen mixtures, being more pronounced when the pH was 4.5 to 5 and less pronounced at pH 7. At the higher pH, more *salt* is precipitated from the solution. It is more desirable to have this take place in vivo through the action of body fluids, and it was for this reason that citric acid was added to the formula.

Basic Compound	Catalytic Agent	Polymerization	Animal Toxicity Reaction	Consistency of Mass	Stability at Fracture Site	Durability of Mass
Elvanol	10% aqueous solution only—none	—	Toxic to 250 Gm. rats in 5-cc. dose	—	—	—
Sodium polyacrylate	Heat and cold	Burns—too much heat for tissues	Nontoxic	Hard	Stable	Indefinite
Elvacet	Acetate emulsion 55% of solid Elvanol	Good	None	Firm—held fairly well	Cement-like	3 months to indefinite time
#370	None	—	Extremely toxic	—	—	—
Elvacet	5% calcium phosphate	Good	None	Firm—but did not hold bone ends together	Not strong enough	3 weeks to indefinite time
#369	None (vehicle 15%)	Poor	None	Soft	—	—
#385A	None (olive-oil solution)	Poor	None	Soft	—	—
#384	None (olive-oil solution)	Poor	None	Soft	—	—
Acrylic polymer 10% compound	10% calcium gluconate	Good	None	—	—	—
10% acrylic polymer compound	10% ferric sulfate 10% aluminum chloride	—	Yes	—	—	—
10% acrylic polymer compound	Iron peptonate in glycerophosphate 6374	—	Yes	—	—	—
10% acrylic polymer compound	6374 and dextran	Good	Very toxic	Hard	—	—
#370	Dextran and glycerin	None	Mild	None	Necrosis	—
Kelcosol 2 Gm to 87 ml	Calcium carbonate 0.3 Gm —citric acid 0.4 Gm H_2O—10 ml	Good	None	Fairly hard	Held bone ends flexible	10 days
Kelmar 3 Gm to 10 ml water	Calcium lactate Calcium carbonate Strontium carbonate	Good	None	Firmest mass of all	Fairly well	2 to 3 weeks
Vegum	5% formaldehyde	Fair	Yes—mild	Firm	Absorbed	3 days
Gelatin	Formaldehyde	Poor	Yes—severe	None	Liquefied	1 day

#370—Polyvinyl methyl ether maleic anhydride copolymer
Acrylic polymer compound (#69931B) is not sodium polyacrylate.
Elvacet polyvinyl acetate emulsion
Glycerin as a catalytic agent proved to be too toxic

#369 = Glycerin glycol dioctyl thylate
#385A = Elvacet 55% (solid) with glycerin
#384 = Elvacet plus #369
6374 = 12% benzyl alcohol with sesame oil G S_2 10M

Gelatin	10% sterile salt solution Calcium carbonate 10 Gm. Magnesium oxide 0.5 Gm. Strontium carbonate 0 5 Gm. Phosphoric acid 4 Gm. Citric acid 4 Gm. pH adjusts as formaldehyde	Good	None Controlled the amount of formaldehyde	Firm	Solid Slowly absorbed over 3 weeks	2 to 3 weeks
Beef collagen with 0.1% acetic acid	Same as gelatin above	Good	Same	Firm	Same	Same

Various experiments were done in order to make these compounds that have been listed, and they are being recorded here as these materials are not readily available for use. For example, with Elvanol, which is polyvinyl alcohol, it was necessary to make numerous experiments to produce an injectable solution. The following formula is the only one which has proved to be satisfactory: Elvanol 10 per cent, glycerin 30 per cent, water 70 per cent. Compound Nos. 369, 385A and 384 were water, alcohol, propylene glycol or dioctyl thylate. For this, a homogenous suspension was produced with olive oil 15 per cent and 20 per cent, rubbing it very hard in the mortar.

In order to use acrylic polymer No. 69931B, which we call acrylic polymer compound, to produce a satisfactory formula for the transparent gel, 10 per cent calcium gluconate and 10 per cent acrylic polymer were used. The mortar and the pestle had to be

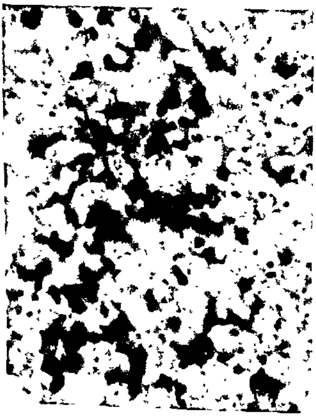


FIG. 9. Ingrowth of fibroblasts in the capillaries and the formation of macrophage about calcium spicules.

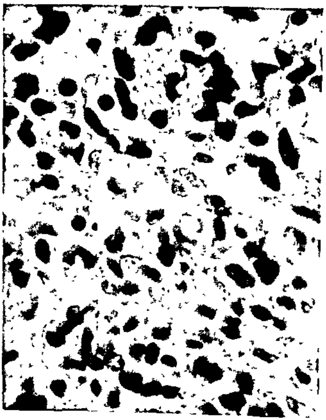


FIG. 10. The same type of change, but suggesting that the macrophage is the precursor of the fibroblast and the chondroblast and the fibroclast and the chondroclast in the formation of new bone.



FIG. 11. Calcium and strontium plastic in fracture with excess callus formation.



FIG. 12. Fresh fracture with a *gelatin* type of plastic injected.



FIG. 13. Healing stage of Figure 12.

warmed thoroughly, the calcium gluconate solution likewise was warmed and *rubbed* gently, small amounts at a time, until a clear, *transparent gel was formed, the mortar being kept* in a hot-water bath during the manipulation.

A 20 per cent solution of acrylic polymer was much too thick to be used in injection.



FIG. 14. Healed stage of Figure 12 at 3 weeks.

Polyvinyl methyl ether maleic anhydride copolymer 10 per cent mixed with propylene glycol 30 per cent and water 60 per cent, and mixed gently while the mortar was in the hot-water bath, would form a clear, firm gel in vitro, which suggested its use in the above experiment. Efforts were unsuccessful to make a 5 per cent acrylic polymer set with 10 per cent iron and ammonium citrate, as probably no ironization took place.

CONCLUSION

The Irish moss solution with the salt brought together the original successful cold polymer, and it was with these cellulose compounds that the author first experimented in 1934. With this vast background it was possible to make up the solutions as outlined with the cellulose plastic, as found in Irish moss, and produce a reasonably hard plastic that was injectable, formed a firm mass and eventually became hydroscopic. These were *in situ* as much as 3 months following the injection at the fracture site in a rat. However, the mass did not become sufficiently firm and did not have a strong enough grasp of the bone ends to achieve a useful gel for a finished product. The research program still continues in the laboratory. The combined use of gelatin and cellulose compounds to prepare a formula for such a plastic now is under investigation in chemistry and the animal laboratories.

BIBLIOGRAPHY

Charry, R.: Plastic reconstruction of hip with nylon, J. Internat. Coll. Surgeons 19:1-23, 1953.

Cobey, M. C.: Plastic immobilization: a new method, Am. Surgeon 20:664-668, 1954.

———: Rest and protection treatment of anterior poliomyelitis, M. Ann. District of Columbia 9:275-276, 1940.

———: Self-illuminating patella retractor, J. Bone & Joint Surg. 22:1103, 1940.

Fleck, Harry R.: Plastics—Scientific and Technological, ed. 3, London, Temple Press, 1951.

Ingraham, Alexander E., Jr., and Matson, D. D.: Medical progress: synthetic plastic materials in surgery, New England J. Med. 236:362, 368, 1947.

Kuhns, John A.: Nylon arthroplasty of the knee, Surg., Gynec. & Obst. 91:351-362, 1950.

Lichter, Alexander: Alloplastic bone graft used experimentally, J. Internat. Coll. Surgeons 22: 238, 1954.

Le Cerca de un Polymerisante Plastico Non-allergic

Summario in Interlingua

Es presentate le description de un projecto de recerca visante al discoperta de un substantia adhesive que esserea tolerate per le corpore animal. Usque nunc nulle plastico satisfacente es disponibile. In concretar se intra le corpore, un grande numero de plasticos produce un tal quantitate de calor que le texitos non pote suffrer lo sin esser destruite. Altere plasticos, que non ha iste defecto calorigene, es nimis caustic e destrue le texitos per lor causticitate. Usque nunc nulle material ha essite inventate que ha un adhesivitate satis forte con texitos organic pro connecter per exemplo duo extremitates ossee e que se concreta intra le texito satis fortemente pro supportar tal extremitates pro un apparato de fixation interne.

Assi il se tracta hic non de conclusiones final sed plus tosto del description del materiales usque nunc studiate e del ideas usque nunc formulate. Nos spera que iste presentation va stimular recercas additional per alteros. Futur reportos in *Clinical Orthopaedics* va monstrar le progressos de nostre investigation.

20

Muscular Dystrophy*

JOHN S. THIEMEYER, JR., A.B., M.D., F.A.C.S.†

In recent years the lifespan of patients suffering from chronic debilitating diseases has increased steadily. This may be due in part to the advent of potent antibiotics, but the improved economic status with its attendant higher nutritional and public health standards also may have contributed to it. In any event, the physician is called upon to care for more and more patients with diseases formerly considered to be rare, and public attention is being focused upon these diseases and demanding cures. Such a disease is muscular dystrophy. Not many years ago it was believed to be a disease of childhood, with death almost inevitable by adolescence. Now it is known to be a disease that also affects the older age groups, and, in addition, it is not uncommon for those children affected at an early age to survive well beyond puberty.

Several years ago the Tidewater Muscular Dystrophy Clinic was established to care for the ever-increasing number of muscular dystrophy patients in the Tidewater Area of Virginia. At present, a total of 17 patients are under direct care, and there are perhaps 10 other cases in the area who have not sought care A review of these patients reveals that the oldest is 50 years of age, and 10 are over 17 years of age. Eleven patients developed the disease in childhood, and 6 first noticed symptoms of muscular dystrophy at or after adolescence.

A complete and up-to-date classification of the types of muscular dystrophy is the following:

Childhood: (1) pseudohypertrophic (Duchenne); (2) nonpseudohypertrophic (Ménière); (3) Juvenile Type of Erb.

Adult: (1) fascioscapulohumeral (Landouzy-Déjerine); (2) menopausal dystrophy; (3) fascioscapular.

A chart of some of the patients' statistics is seen on pages 215 to 217.

It has been observed that the progress of the disease is proportional to the age of onset; thus, the earlier it appears, the more rapid and the more sinister its course. The differential diagnosis is not difficult, but it may be confused with the following:

1. Dermatomyositis, which usually displays skin lesions and calcific plaques, and responds to ACTH and Cortisone while muscular dystrophy does not.
2. Acute myositis, which usually is localized to 1 muscle or muscle group, and does not display hypertrophy of any degree but usually is accompanied by edema.
3. Scleroderma, which does not display muscle hypertrophy, pathologic muscle changes, or creatinuria, as in dystrophy and usually shows calcinosis.
4. Amyotonia congenita, which does not show hypertrophy, appears earlier in life, and does not show creatinuria.
5. Myotonia dystrophia, which usually shows lens opacity and does not undergo hypertrophic phase.
6. Charcot-Marie-Tooth disease, which

---

* Presented at the 1955 meeting of the Association of Bone and Joint Surgeons

† Norfolk, Va.



FIG. 1. Pseudohypertrophic type (Duchenne)



FIG. 2. Nonpseudohypertrophic type (Ménière)

shows marked atrophy of peroneal muscle group and no hypertrophy or creatinuria.

7. Multiple sclerosis, which does not display creatinuria or muscle hypertrophy, but shows neurologic changes not seen in dystrophy.

The final diagnosis usually lies in the pathologic changes seen in the muscle biopsy and the urine, which display persistent increase of creatine and creatinine.

Treatment of the disease has not changed much in recent years. However, it has been shown that proper and persistent physiotherapy can prevent contractures and thus impede the loss of function of associated musculature and progressive disability. The hands usually are the last parts to be involved, so that therapy and training should be directed to work with the hands, looking toward the maximum productivity of the patient. Vocational and occupational training is vital conjunctive therapy, as these patients often can be kept productive members of society for many years.

Drug therapy has been as diversified in this disease as in any other, but, in the author's opinion, only 2 drugs are of any value at this time. They are alpha-tocopherol or vitamin E and amino-acetic acid.

Investigations indicate that amino-acetic acid supplies a deficiency in muscle metabolism that exists in muscular dystrophy. Patients are unable to *fix* or retain creatine in the muscles and experience gradual loss of



FIG. 3. Fascioscapulo humeral type (Landouzy-Déjerine)

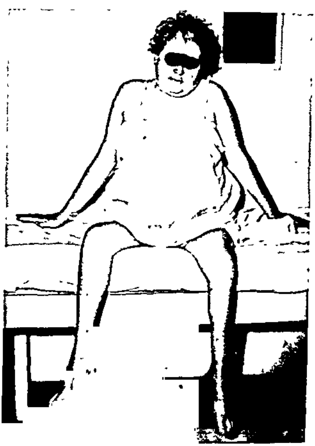


FIG. 4. Menopausal dystrophy

muscle function. Feeding creatine as such does little good, as most of it passes through the body and is excreted promptly in the urine. If amino-acetic acid is given, however, creatine may be formed, which, though at first excreted so that the urine shows even greater amounts of creatine and creatinine, may later be retained in the muscles and become available for utilization in muscle contraction. Treatment has to be continued, because amino-acetic acid supplies a deficiency rather than effects a cure. In addition, laboratory studies have shown that deficiency of vitamin E can produce pathologic changes of muscle indistinguishable from those found in muscular dystrophy, and when vitamin E is supplied this condition can be reversed. It is seen clinically that patients do better with than without vitamin E.

The patients in this group have all been on the drugs mentioned. The dosage of alpha-tocopherol varied from 100 to 300 mg. per day, according to age. Glycocoll was given in dosage from 10 to 15 Gm. daily, according to age. It was found that doses over 15 Gm. usually caused urinary distress, such as frequency and burning. This therapy has been continued for about 3 years.

Of the 17 patients, 3 have had inadequate follow-up. Of the remaining 14, 4 improved, 6 showed no change, and 4 declined. Ten out of 14 have remained productive. As funds were limited, extensive muscle-function tests could not be done However, each patient was seen by the author at about monthly intervals, and his evaluation was used as a measure of progress or decline.

Financial assistance of some type usually is required by these patients, as drugs and therapy are expensive and continual, and few people can afford them unaided for the long periods required.

About a year and a half ago a report originated from the University of California by Dr. Van Meter that outstanding results were being obtained in muscular dystrophy

Muscular Dystrophy Cases

Name and Type	Age When First Seen	Age of Onset	Medication	Progress and Pertinent Data
B. V. White male Pseudohypertrophic	5	3 yr.	Alpha-tocopherol 100-300 mg. /day, glycocoll, physiotherapy, foot braces, 1953. Corset, 1955.	Positive biopsy, 1951. Unknown febrile illness at 2 yr. Pectoral atrophy and hypertrophy of calves. Total creatinine 317.8 mg./410 cc. Positive *slip through* and *push up* signs. No change.
F. V. White male Pseudohypertrophic	6	3½ yr.	Alpha-tocopherol 300 mg. Glycocoll 10 Gm./day, 1953. Night splints, 1954.	Poor concentration and speech. 1947—cerebral hemorrhage following prolonged difficult labor. Pectoral atrophy and hypertrophy of calves. Muscle biopsy positive, 1952. Positive *slip through* and *push up* signs. Total creatinine 416 mg./320 cc. Preformed creatinine 236.8 mg./320 cc. Creatine 144.2 mg./320 cc. Progressive decline.
J B. White male Pseudohypertrophic	7	1½ yr.	Alpha-tocopherol 100 mg./day, glycocoll 5-10 Gm./day, exercises, 1953. Protein hydrolysate and rubrafolin, 1954. Decline. Resumed glycocoll 10 Gm./day and alpha-tocopherol 200 mg /day, 1954. Glycocoll 30 Gm./day, 1955.	Usual childhood diseases. Pectoral atrophy. Calf hypertrophy. Biopsy 1950 positive. Good family care. No contractures.. Total creatinine 564.3 mg./510 cc. Preformed creatinine 353.4 mg./570 cc. Creatine 152.1 mg./570 cc. Sister (twin) with heart condition and frequent colds, other sister normal. Positive *push up* and *slip through* signs. No change.
J. M. White male Pseudohypertrophic	8	6½ yr.	Glycocoll 5-10 Gm./day, alpha-tocopherol 200-300 mg /day, 1953. Physiotherapy, 1954.	Poor pectorals, hypertrophy calves. *slip through* and *push up* signs positive. No contractures. One of 3 brothers with the disease. No change.
M. M. White male Nonpseudo-hypertrophic	9	6 yr.	Glycocoll 5 Gm /day. Alpha-tocopherol 200 mg./day. Wedge casts (1951) physiotherapy. Glycocoll 10 Gm./day. Alpha-tocopherol 300 units/day, 1954.	Marked atrophy of arms and legs; contractures knees, hips and feet. One of 3 brothers with disease. Contractures corrected partially. No change.
R. B. White male Pseudohypertrophic	9	3 yr.	Glycocoll 10 Gm./day at Johns Hopkins, 1946. Glycocoll 5-10 Gm./day, 1953. Alpha-tocopherol 100-300 mg./day, 1954. Stretching, leg braces added 1954 with above. Rubrafolin 1 capsule/day and protein hydrolysate 30 Gm./day for 1 year with decline.	Pectoral atrophy. Calf hypertrophy, marked equinovarus deformity of both feet with calf contractures. *Push up* and *slip through* signs positive. Total Creatinine 713.4 mg. /820 cc. Preformed creatinine 287 mg./820 cc. Creatine 454.6 mg./820 cc. Progressive decline.
H. H. Nonpseudohypertrophic	13	8 yr.	Glycocoll 10 Gm./day. Alpha-tocopherol 300 units/day. Ankle braces.	Cephalohematoma at birth. Pectoral atrophy. Calf atrophy with minimal contracture. No change.

NAME AND TYPE	AGE WHEN FIRST SEEN	AGE OF ONSET	MEDICATION	PROGRESS AND PERTINENT DATA
W. F. Negro male Nonpseudohypertrophic	17	5 yr.	Glycocoll 5-10 Gm./day, 1953. Alpha-tocopherol 200 mg./day. Physiotherapy. Long leg brace, right, and crutches.	Triple arthrodesis and heel-cord lengthening at 7 yr. Pectoral atrophy. Calf atrophy. Contractures, hips, knees and ankles. Lumbar lordosis. Ambulatory by using hands on thighs. *Push up* and *slip through* signs positive. Total creatinine 606.8 mg./820 cc creatinine 1148 mg./820 cc. Preformed creatinine 606.8 mg./820 cc. Creatine 627.8 mg /820 cc. No change.
L. B. White male Pseudohypertrophic	17	10 yr.	Glycocoll 10 Gm./day. Alpha-tocopherol 300 mg /day. Long leg extension braces, 1953. Rubrafolin 1 capsule b i.d. Protein hydrolysate 5 Gm. t.i.d., 1954. No change.	Atrophy pectorals, biceps and triceps. Atrophy legs with marked contracture. Late pseudohypertrophic. About in wheel chair. One of 2 brothers with the disease Total creatinine 819 mg/700 cc. Preformed creatinine 504/700 cc. Creatine 365.4/700 cc. Improved.
D. B. White male Pseudohypertrophic	19	10 yr.	Glycocoll 10-20 Gm./day. *Alpha-tocopherol 300* mg/day. Long leg extension braces. Physiotherapy.	Atrophy pectorals and legs with contractures. *Wheel* chair. Total creatinine 747.5/575 cc. Preformed creatinine 500 3/575 cc. Creatine 286.7 /575 cc. One of 2 brothers with disease. Improved.
R. S. White male Fascio- *scapulohumeral*	24	21-22	Glycocoll 10 Gm./day and alpha-tocopherol 300 mg./day, 1954. Physiotherapy. *Rubrafolin and protein hy-*drolysate 15 Gm./day, 1954. Alpha-tocopherol 300 mg./day and glycocoll 15 Gm./day, 1955.	Tapir facies, atrophy shoulder girdle. Was in Navy and retired 1952 with the disease *after biopsy and urine studies.* Employed gainfully. Improved.
H. F. White male Fascio- scapulohumeral	30	14 yr.	Alpha-tocopherol 300 mg./day. Glycocoll 10 Gm./day. 1954.	Biopsy 1943 positive. Shoulder atrophy and tapir facies. No follow-up.
C. H. White male Fascio- scapulohumeral	35	17 yr.	Alpha-tocopherol 300 mg./day and glycocoll 10 Gm./day. Foot brace, right, 1954.	Was in National Guard. Had mastoiditis and progressive deafness as child. Marked shoulder atrophy. Tapir facies. Employed gainfully. Improved.
E. B. White female Fascio- scapulohumeral	37	18 yr.	Alpha-tocopherol 300 mg./day and glycocoll 10 Gm./day, 1953. Wheel chair, 1953. Corset. Rubrafolin 2 capsules/day. Protein hydrolysate 30 Gm., 1954. Slight improvement after return to alpha-tocopherol and glycocoll.	Girdle atrophy, leg weakness. About with support. Two children. Total creatinine 1495.5 mg./1372 cc. Preformed creatinine 621.1 mg. /1372 cc. Creatine 1002.7 mg /1372 cc. *No change.*

D. J. Negro female Menopausal dystrophy	40	38 yr.	Glycocoll 10 Gm./day, alpha-tocopherol 300 Gm./day. Corset, physiotherapy. Wheel chair.	Biopsy positive, 1953. Pectoral atrophy, generalized weakness. Shuffling gait, requires support for ambulation. Total creatinine 1050 mg./785 cc. Preformed creatinine 600 mg. /785 cc. Creatine 500 mg./ 785 cc. Progressive decline.
E. B. White female Menopausal dystrophy	50	47 yr.	Glycocoll 10 Gm./day, alpha-tocopherol 300 mg./day. Wheel chair.	Severe weakness of both legs and arms, requiring wheel chair. Progressive decline.

by the use of Rubrafolin and protein hydrolysate. A group of 5 of our patients, therefore, were placed on this regimen. Of this group, 3 demonstrated no change in a period of 1 year and 2 declined considerably. Our studies, therefore, do not seem to support the findings of Dr. Van Meter.

To summarize, it is believed that muscular dystrophy is becoming progressively prominent, and that proper therapy applied consistently and continuously can prolong greatly the productivity and the happiness of the patient suffering from this disease.

Dystrophia Muscular

Summario in Interlingua

In recente annos il ha habite un augmento del numero de patientes con dystrophia muscular, principalmente a causa del augmentate superviventia de patientes con iste morbo. Isto se debe in parte al advento de potente antibioticos e forsan al meliorate stato economic. In ultra, le classification de dystrophia muscular es devenite plus inclusive. Le sequente es un classification de dystrophia muscular in forma currente.

A. Juveniles
   1. Pseudohypertrophic (Duchenne)
   2. Non-Pseudohypertrophic (Ménière; Typo Juvenil de Erb)

B. Adultos
   1. *Facio-Scapulo-Humeral* (Landousy-Déjerine)
   2. Menopausal
   3. *Facio-Scapular.*

Le morbo debe esser differentiate ab dermatomyositis, acute myositis, scleroderma, amyotonia congenite, myotonia atrophic, morbo del typo Charcot-Marie-Tooth, e sclerosis multiple. Le diagnose es possible solmente per biopsia muscular e tests urinari pro creatina e creatinina. Le tractamento del morbo non ha cambiate in recente annos. On crede que le sol drogas de valor que es nunc disponibile es alpha-tocopherol o vitamina E e acido aminoacetic. Iste drogas—conjunctemente con therapia physic e varie formas de therapia supportative—esseva usate in un serie de 17 patientes Ex le total, 4 monstrava melioration, 6 remaneva incambiate, 4 se deteriova, e 3 escappava a adequate observation. Assi 10 individuos in un gruppo de 14 patientes con dystrophia muscular poteva continuar un existentia productive. Le drogas rubofolina e hydrolysato de proteina esseva essayate a causa del excellente resultatos reportate pro illos per un gruppo de investigatores. Il se monstrava que illos esseva sin valor. Nos opina que medication e rehabilitation pote adjutar un bon procentage de patientes con dystrophia muscular a remaner productive pro un longe periodo de lor vita.

21

Calcification of the Intervertebral Disk: Disappearing, Dormant and Silent*

A. M. RECHTMAN, M.D., M. B. HERMEL, M.D., S. M. ALBERT, M.D., AND A. G. BOREADIS, M.D.

Three cases of idiopathic calcification of the intervertebral disk are presented. They were observed and studied in conjunction with 18 previously recorded. This stimulated further clinical investigation as follows: (1) classification of this form of disk calcification; (2) discussion concerning etiology; (3) review of the anatomy, the physiology and the pathology of the tissues involved to determine why in some individuals there are objective findings as well as pain and other complaints, while in others the disturbance is *silent;* and (4) stimulation of research interest, which may give a definitive answer to the etiology and add to the knowledge of the disk problem in general.

The idiopathic type of disk calcification is subdivided by the authors into the *disappearing,* the *dormant* and the *silent* forms.

In the disappearing form, the onset is associated with acute symptoms. Roentgen study shows a calcific deposit in the disk, and, after a period, often of some months or longer, further roentgen study shows that the calcification is disappearing. This group comprises 12 cases.

In the dormant form, the calcific deposit was observed as an incidental finding; however, acute symptoms occurred subsequently. Later roentgen studies showed that the lime deposits disappeared. There were 2 such cases.

The term *silent disk calcification* was ascribed to those in whom the disk deposit was noted as an incidental finding on the roentgen films, and there were no other associated signs or symptoms. There were 4 of these cases.

Three cases lacked sufficient data for classification. The several forms or subclassifications are not necessarily clear cut, but they may have some factors in common.

CASE REPORTS

Case 19. A 3-year-old white girl was well until she awakened, several times, on October 20, 1951, and complained of "backache." Examination 2 days later showed a well-developed child who had a *left torticollis. She raised* and shifted the eyes, but maintained a relatively fixed attitude of the head and the neck. She did not permit extension, lateral bending or rotation of the head and the neck to the right, but the other motions were free. There were no other untoward objective findings and no pain or other subjective complaints. Detailed study of the upper extremities was negative. The temperature was normal. Roentgen studies revealed a disk-shaped calcific deposit in the region of the nucleus pulposus of the 4th cervical intervertebral disk (Fig. 1).

The patient was admitted to the Albert Einstein Medical Center, Northern Division, Philadelphia, on October 22, 1951. A head halter was applied.

Examination 1 day after the child was in

* From the Departm[illegible] of Orthopedic Surgery and Radiology, Albe[illegible] nstein Medical Center, Northern Division, Ph[illegible]delphia, Pa.

traction showed that the head and the neck were held in neutral attitude. The torticollis was corrected, and motions were possible through a normal range.

Examination 2 days later again was entirely negative. However, because the initial films suggested subluxation between C-2 and 3, and because the cervical curve was flat, a Calot plaster collar jacket was applied prior to hospital discharge.

The plaster was removed after 5 weeks, and all motions of the head and the neck were normal. Roentgen studies at this time revealed a normal cervical curve. There was no restriction of movement on flexion and extension studies; also, there was no change in the appearance of the calcific shadow in the 4th cervical disk. The laboratory reports showed a normal blood count, urinalysis and sedimentation rate. Repeated clinical studies were negative.

Another roentgen study was not made for 27 months (March 10, 1954); it showed almost complete disappearance of the calcifications (Fig. 2).



Fig. 1. Right posterior oblique and right lateral projections of cervical spine disclose an oval-shaped calcification in the nucleus pulposus of the intervertebral disk between C 4 and 5. Film was made during acute stage of illness, and cervical lordosis is straightened.

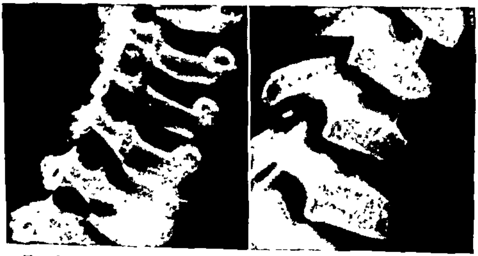


Fig. 2. Re-examination of same areas of cervical spine 27 months later, enlarged photographically, disclose practically complete disappearance of calcification. Only 2 minute flecks of calcification are seen in the lateral view.

Case 20. A 37-year-old male, a cellist, was well until late April, 1953, when he noted intermittent "jabbing pain, just like a flash," at the dorsolumbar region; lesser annoyance extended to the shoulder blades. This continued for 3 days. A few days previously he had felt chilly, but his teeth did not chatter; his temperature had not been taken, but he does not believe that he had a fever. He did not feel sick, and played at a concert that evening. Three days after the onset he had "constant pain," also on occasion a severe constricting sensation about the anterior lower right rib margin, which would abate within a half minute. The constricting sensations occurred innumerable times during a period of 4 days. He was unable to work during this time. He slept poorly and "actually had to crawl out of bed in the morning."

About a week after the onset, point tenderness was noted at the spinous process of the 7th thoracic vertebra. The paraspinal muscles were in spasm. The trunk was flexed moderately, as this was a position of comfort. Motions of the trunk aggravated the pain, as did getting into and out of a car and on the x-ray table. However, the discomfort was primarily in the lower dorsal area and the anterior lower right rib cage; the neck felt stiff.

Roentgen studies about a week after the onset of symptoms revealed several amorphous calcific plaques, centrally placed, in the 7th dorsal intervertebral disk (Fig. 3). This was the site of tenderness to pressure on clinical examination.

The patient was given roentgen therapy, 100 r on 3 consecutive days. He was improving prior to this treatment, so that he questioned if it helped. His improvement continued over a period of 2 weeks, at which time the pain had abated. Motion in the back was free and painless, but a subjective sense of stiffness in the neck persisted "for a good while." It was "a couple of weeks" before he could get into and out of a car with ease. However, 5 days after the initial onset he was able to work "without too much annoyance." The "spasms," anterior lower right chest, abated before the roentgen treatment was started. Within a few weeks the back, the chest and the neck were entirely well, and he has had no trouble since.

Roentgen study of the dorsal spine after 7½ months showed a marked decrease in the size of the calcification.

The past history was irrelevant. The only other orthopaedic disturbance was a "toothachy type" of pain in both shoulders, especially the right, which the patient first noted in 1949. Between 1949 and 1953 the patient had shoulder annoyance about twice a month, never severe, lasting for about a day, and related to "how busy I was with the cello." During the past month, however, the right shoulder had been asymptomatic. He had an acute exacerbation of the right shoulder pain about 1 week following the onset of his back annoyance. The shoul-

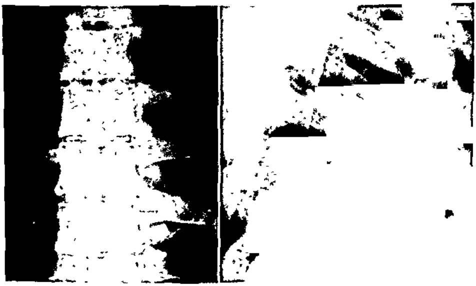


FIG. 3. Anteroposterior and lateral roentgenograms of mid-dorsal spine showing extensive amorphous calcification in the region of the nucleus pulposus of the 7th dorsal intervertebral disk. Films were made during the acute phase of the illness.

der was roentgenographed on May 11, a week after the back was studied, because of recurrence of pain. Roentgen treatment was begun May 12, 1953. The films of the right shoulder showed an extensive irregular amorphous calcific deposit adjacent to the greater tuberosity. The roentgen appearance was not that of a recent lesion. Films of the cervical spine were negative.

A report of a roentgen study of the dorsal spine on May 2, 1949, 4 years before the onset of his current back trouble, stated that the patient had a calcific deposit in the intervertebral space of the 7th dorsal. These films were not available for comparison with the more recent studies. They had been made at the request of a chiropractor. The patient's complaint then was "long-standing migraine headaches." He had no backache. The calcific shadow in the 7th intervertebral disk, noted at that time, might therefore be considered a fortuitous incidental finding.

The history and the orthopaedic examination were made 11 months after the onset of his back pain. Study showed a 37-year-old male with fairly good posture. There was no spasm of the paraspinal muscles. All motions of the head, the neck and the trunk were possible through a normal range of movement, both actively and passively. Studies of the upper extremities also showed a normal and equal range of active and passive movement, except for mild restriction on rotating internally the right upper extremity behind the back. Films at this time showed slightly further disappearance of the calcific shadow (Fig. 4).

Case 21. A male, aged 5 years. (We are indebted to Dr. Mark Harwood,[4] of Syracuse, New York, for the details and the roentgen studies in this case.)

Two weeks prior to the current episode, the child had "infected tonsils and a cervical adenitis." His highest temperature was 102° F. Apparently he recovered and returned to school. Five days later, November 15, 1952, the child was tumbling and "trying to stand on his head." That evening he complained of a stiff neck, pain on the right side of the head, and a burning sensation on the right side of the throat. He had no medical care for 5 days. Study then showed considerable paraspinal cervical muscle spasm and a left torticollis. Motions of the head and the neck were limited, especially in flexion and lateral bending to the right. There was a flattening of the left side of the face and skull, asso-

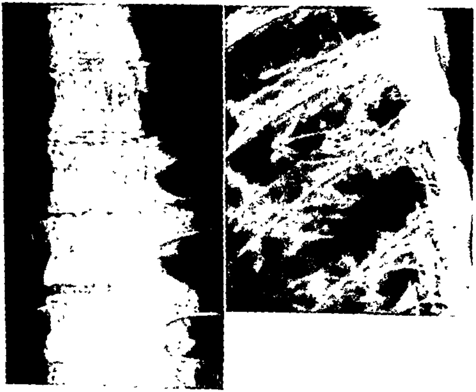


FIG. 4. Follow-up roentgen films made 11 months later reveal marked diminution in size of the calcific deposit with only a small residual calcification being visible at the posterior aspect of the intervertebral space.

ciated with antecedent torticollis. Head halter traction was applied. The patient improved rapidly, and left the hospital within a week, wearing a felt cervical collar.

The child was seen on occasion for 4 months. During this period he had no subjective complaints; the range of motion in the neck was normal.

The initial roentgenograms, made the following day, showed that the cervical curve was flattened. There was a linear amorphous calcification, centrally placed, in the 4th cervical intervertebral disk (Fig. 5).

Laboratory studies were made on November 18 and 24, 1952. The sedimentation rate was zero. Tuberculin reaction in dilution 1:1000 was negative, as was the blood Wassermann test. A throat culture showed no growth. The white and the differential blood counts on November 18 and 24 were normal.

Roentgen re-examination 21 months later disclosed that the calcification had disappeared except for 2 pinhead-sized calcific flakes (Fig. 6).

Discussion

Case 19 may be classified as the *disappearing form* of disk calcification. The child had a torticollis. The site of reversal of the cervical curve coincided with that of the ap-



FIG. 5. Anteroposterior and lateral views of cervical spine made during height of clinical picture disclosed a flat calcific deposit in the nucleus pulposus of the 4th cervical disk. The cervical lordosis is flattened.



FIG. 6 Repeat study of cervical spine 21 months later shows almost complete reabsorption of calcific deposit. Only 2 small flecks remain, as seen in the anteroposterior view

parent subluxation, C-2 and 3 shown on the initial roentgenograms. Silverman[20] states that roentgen examination, made while the head is held rigid, may lead to an erroneous diagnosis of subluxation of the cervical spine. In addition, the rotary changes associated with torticollis may also contribute toward the appearance of subluxation. A calcific deposit was noted in the 4th cervical disk. Irritation from this may well have been the stimulating factor that caused muscle spasm and the roentgen appearance of subluxation. At least 7 of the 9 cases with cervical involvement had torticollis; the other 2 complained of "stiff neck."

Case 20 was classified as the *dormant form* of disk calcification. The prime difference in this case from the other 2 described by us, as well as those reported by previous writers, was the fortuitous incidental finding of a dormant intervertebral disk calcification (in 1949) 4 years before the onset of back pain. Four years later the patient had acute symptoms, and 10 months later the calcification had disappeared almost entirely. This patient also had a chronic calcific tendinitis of the rotator cuff of the right shoulder, with intermittent mild episodes of annoyance for 4 years, and an acute exacerbation within a week following his back episode in 1953. He was the only adult in our series, and the third in a total of 21 cases. He was treated with roentgen therapy, but was improving before it was started, and he could not ascribe his recovery to this modality.

Case 21 is another example of the *disappearing form* of idiopathic calcification. The child presumably had recovered from the effects of tonsillitis and cervical adenitis of early November, 1952, as evidenced by 2 negative white and differential counts, normal temperature readings and zero sedimentation rates. The calcification in the disk, with acute symptoms on November 15, therefore, may cause one to question if there was any relationship to the upper respiratory illness of November 1.

CLINICAL FEATURES

Table 1 summarizes the important clinical findings of the 18 cases previously reported and the 3 that we are adding. Twelve of the children were under the age of 10; 6 were between the ages of 10 and 12. The youngest child was 6 months of age. Of the 21 cases, 18 were in children. At least 10 of the children had fever, but probably not all on a basis of calcification in the disk alone. All the patients had pain, mild to severe, except the 7 who were roentgenographed for disturbances other than the spine and in whom the calcification was discovered as an incidental finding. Most of the patients complained of a stiff neck or back. Tenderness over the involved portion of the spine usually was elicited in those cases in which this factor was discussed. Roentgen studies disclosed characteristic calcifications in the region of the nucleus pulposus of the involved disks.

Table 2: Fate of the Calcification. The details of this study are well illustrated in Table 2. In 1 (Case 20), the calcification was noted as an incidental finding in 1949. Four years later, however, following the onset of acute symptoms, subsequent roentgen study showed the disappearance of the calcific deposit after 10 months. In Case No. 6, the calcification was also noted as an incidental finding. Five months later, the patient had a mild ache; 2 months following that he had acute symptoms in the lower cervical spine, the site of a deposit. A year later the calcific mass had disappeared. These 2 cases are of the dormant type.

Table 3: Disk Involvement. Thirty-seven disks showed calcification. The details concerning the disks affected with calcification are shown in Table 3. It shows the region of the spine affected. It indicates those in whom only 1 disk was involved, those in whom contiguous disks showed calcification, and others in whom the affected disks were separated by 1 or more disks that appeared to be normal on roentgen study.

The lower cervical area, where motion and strain of normal function are greatest,

had but a single involvement in 7 cases. In the dorsal region, the disks involved did not bespeak specifically the sites of strain, which are usually associated with the junction of the relatively fixed thoracic area with the more freely movable cervical or lumbar spine. In only 3 cases was a lumbar disk, the 1st, involved.

Discussion

Calcification in the intervertebral disks first was mentioned by Luschka (1858) in his book on anatomy. Benke showed it in *Roentgen Study in the Cadaver,* 1897, and Calvé and Galland described it in *Man,* 1922 (quoted by Weens[21] and Sandström[16]). Permanent calcification was studied thoroughly by Schmorl[18] and Schmorl and Junghanns.[19] They considered it to be a degenerative change, increasing in frequency with age.

Anatomy: Morphology

The intervertebral disk is formed by different tissues, and calcification may occur in all of them.[15] Anatomically, the disk comprises a thin superior and inferior plate of hyaline cartilage, which may be the site of permanent calcification, just as it may occur in this structure elsewhere in the body. The peripheral ring-shaped annulus fibrosus is composed of fibrocartilage and connective tissues. In addition, at the site of the junction of the nucleus pulposus with the annulus fibrosus, microscopic study shows a structure identical with that found in ordinary tendinous tissue. This structure may be the site of permanent and/or temporary calcification, just as it occurs in such tissue elsewhere in the body, whereas the remainder of the annulus fibrosus may be the site of permanent calcification. On microscopic study, the nucleus pulposus has the same appearance and structure as soft connective tissue seen in peritendinous tissue, and so, similarly, may be the site of either or both temporary and permanent calcification The permanent type of calcification usually involves the annulus fibrosus, although at times the nucleus pulposus also may be affected; whereas the idiopathic type involves primarily the region of the nucleus pulposus.

Etiology

Exactly what causes the formation of lime deposits is not known. It may arise from reaction to noxious agents, resulting in local nutritional disturbance. Calcification probably is solely a local lesion that occurs in devitalized tissue on a physical and chemical basis. Trauma, acute or static, inflammation, an excess or toxic intake of vitamin D, disturbance of calcium or other metabolic disorders, chemicophysical changes, perhaps on a degenerative basis, vascular disease and aseptic necrosis have been postulated by previous authors[1,6,13,14,16,17,18,19,21] as causative factors for calcifications. Degenerative changes may be excluded as the cause of idiopathic calcification, since 18 of the 21 cases occurred in children, and also, because of its temporary nature, rapid improvement and recovery are noted so frequently.

It is our impression that the etiologic role mentioned by previous writers may be only the stimulating factor either to be concerned with calcification and symptoms or to cause a previously dormant calcific process to become acute. Baron,[1] Lyon[12] and Barsony and Koppenstein, quoted by Sandström,[15] consider that the calcifications have no clinical significance. It is our opinion, however, that the calcifications have diagnostic, as well as pathologic, significance.

It was interesting to note that workers in this field whom we contacted, Doctors Neuhauser, Caffey, Hirsch, Friberg, Steindler and others, indicated that they had observed the condition, but that they only considered it to be a curious finding, of unknown significance. Friberg[7] and Hirsch,[8] of the Karolinska Institute Orthopediski Klinik of Stockholm, Sweden, and their co-workers made extensive clinical, roentgen, operative, chemical and other laboratory, as well as autopsy, studies of about 20,000 spines. The former stated that he did not know the cause of

temporary calcification; the latter, "Our biological knowledge is not sufficient to explain why disks in children sometimes calcify. I have put your letter on top of the questions we want to investigate."

While the etiology of idiopathic calcification of the disk is not established definitely, its localization may be explained on a basis of the morphology of the disk. Hirsch and Schajowicz[5] and others have observed that the annulus fibrosus develops during intrauterine life. With growth, it soon develops and becomes firmly attached to the cartilaginous plate and the adjacent rim of the vertebral body. Prior to its full development, but rarely before 15 years of age, oval and irregular areas, crescentic in course, are found between the lamellae. At these sites the normal fibrous structure is in the process of disappearing, or has already disappeared, and the area is filled with a mucilaginous mass.[9] Structurally, the process results in crescentic cracks in the annulus. These are more numerous adjacent to the nucleus pulposus. However, they do not communicate with it, nor do they reach the periphery of the disk. Is it probable that these calcific deposits occur most frequently in children because the disks have not yet developed sufficiently to protect them from irritations that cause the calcifying process? May the crescentic cracks, which rarely appear before the age of 15, then act as a barrier to protect the disks from the irritations that cause idiopathic, often temporary, calcifications that form primarily in the region of the nucleus pulposus?

Blood Supply

In the embryo, vessels from the cancellous portion of the vertebral body, as well as the ventral and the dorsal marginal vessels, run to the cartilaginous plate. These vessels do not penetrate the portion of the disk that later will become the nucleus pulposus or the annulus fibrosus. Hirsch and Schajowicz,[8] quoting Ubermuth, Boehmig and Schajowicz, said, "Thus, the nucleus pulposus and the annulus fibrosus are completely without vessels during every phase of the individual's life." However, they did note branches entering from the perichondrium into the calcified marginal epiphyses (seen when these growth centers begin to ossify at the age of 13), and the vessels disappear when the epiphyses fuse at about 20 years of age. They also noted a few vessels, but only in the outermost layers of the ligaments. Therefore, may it be that the blood supply from the central axial vessels, which runs only to the cartilaginous plate, and the dorsal and the ventral vessels carry toxins, infectious organisms or other noxious materials to interfere temporarily with the nutrition of the disk and its nucleus pulposus?

The bone adjacent to the disk is cancellous primarily; the disturbance usually occurs in children. Both factors are associated with good blood supply. May it not be, therefore, that this is among the probable factors to explain why the pathology is reversible and the calcification temporary?

Nerve Supply: Pain

Pain usually was a prominent symptom, except in the 7 cases in which calcification was noted in the disk as an incidental finding. The disk, however, is devoid of nerve supply, as shown by the studies of Hirsch and Schajowicz.[8] Fairly large bundles of nerve fibers have been observed outside the ventral and the dorsal ligaments, and thinner bundles accompanying the vessels could only be followed into the outermost parts of these ligaments Nerves never have been observed to penetrate the ligaments to their junction with the disk, nor into the annulus.

An attempt will be made to explain the symptom of pain in the *disappearing* and in the later stages of the *dormant* form of disk calcification; also, the absence of this symptom in the earlier stage of the *dormant* and the *silent* forms.

It is suggested that there may be a correlation between certain facets of *peritendinitis calcarea*, an outstanding example of

had but a single involvement in 7 cases. In the dorsal region, the disks involved did not bespeak specifically the sites of strain, which are usually associated with the junction of the relatively fixed thoracic area with the more freely movable cervical or lumbar spine. In only 3 cases was a lumbar disk, the 1st, involved.

Discussion

Calcification in the intervertebral disks first was mentioned by Luschka (1858) in his book on anatomy. Benke showed it in *Roentgen Study in the Cadaver,* 1897, and Calvé and Galland described it in *Man,* 1922 (quoted by Weens[21] and Sandström[16]). Permanent calcification was studied thoroughly by Schmorl[18] and Schmorl and Junghanns.[19] They considered it to be a degenerative change, increasing in frequency with age.

Anatomy: Morphology

The intervertebral disk is formed by different tissues, and calcification may occur in all of them.[15] Anatomically, the disk comprises a thin superior and inferior plate of hyaline cartilage, which may be the site of permanent calcification, just as it may occur in this structure elsewhere in the body. The peripheral ring-shaped annulus fibrosus is composed of fibrocartilage and connective tissues. In addition, at the site of the junction of the nucleus pulposus with the annulus fibrosus, microscopic study shows a structure identical with that found in ordinary tendinous tissue. This structure may be the site of permanent and/or temporary calcification, just as it occurs in such tissue elsewhere in the body, whereas the remainder of the annulus fibrosus may be the site of permanent calcification. On microscopic study, the nucleus pulposus has the same appearance and structure as soft connective tissue seen in peritendinous tissue, and so, similarly, may be the site of either or both temporary and permanent calcification. The permanent type of calcification usually involves the annulus fibrosus, although at times the nucleus pulposus also may be affected; whereas the idiopathic type involves primarily the region of the nucleus pulposus

Etiology

Exactly what causes the formation of lime deposits is not known. It may arise from reaction to noxious agents, resulting in local nutritional disturbance. Calcification probably is solely a local lesion that occurs in devitalized tissue on a physical and chemical basis. Trauma, acute or static, inflammation, an excess or toxic intake of vitamin D, disturbance of calcium or other metabolic disorders, chemicophysical changes, perhaps on a degenerative basis, vascular disease and aseptic necrosis have been postulated by previous authors[1,6,13,14,16,17,18,19,21] as causative factors for calcifications. Degenerative changes may be excluded as the cause of idiopathic calcification, since 18 of the 21 cases occurred in children, and also, because of its temporary nature, rapid improvement and recovery are noted so frequently.

It is our impression that the etiologic role mentioned by previous writers may be only the stimulating factor either to be concerned with calcification and symptoms or to cause a previously dormant calcific process to become acute. Baron,[1] Lyon[12] and Barsony and Koppenstein, quoted by Sandström,[15] consider that the calcifications have no clinical significance. It is our opinion, however, that the calcifications have diagnostic, as well as pathologic, significance.

It was interesting to note that workers in this field whom we contacted, Doctors Neuhauser, Caffey, Hirsch, Friberg, Steindler and others, indicated that they had observed the condition, but that they only considered it to be a curious finding, of unknown significance. Friberg[3] and Hirsch,[6] of the Karolinska Institute Orthopediski Klinik of Stockholm, Sweden, and their co-workers made extensive clinical, roentgen, operative, chemical and other laboratory, as well as autopsy, studies of about 20,000 spines. The former stated that he did not know the cause of

temporary calcification; the latter, "Our biological knowledge is not sufficient to explain why disks in children sometimes calcify. I have put your letter on top of the questions we want to investigate."

While the etiology of idiopathic calcification of the disk is not established definitely, its localization may be explained on a basis of the morphology of the disk. Hirsch and Schajowicz[8] and others have observed that the annulus fibrosus develops during intrauterine life. With growth, it soon develops and becomes firmly attached to the cartilaginous plate and the adjacent rim of the vertebral body. Prior to its full development, but rarely before 15 years of age, oval and irregular areas, crescentic in course, are found between the lamellae. At these sites the normal fibrous structure is in the process of disappearing, or has already disappeared, and the area is filled with a mucilaginous mass.[9] Structurally, the process results in crescentic cracks in the annulus. These are more numerous adjacent to the nucleus pulposus. However, they do not communicate with it, nor do they reach the periphery of the disk. Is it probable that these calcific deposits occur most frequently in children because the disks have not yet developed sufficiently to protect them from irritations that cause the calcifying process? May the crescentic cracks, which rarely appear before the age of 15, then act as a barrier to protect the disks from the irritations that cause idiopathic, often temporary, calcifications that form primarily in the region of the nucleus pulposus?

Blood Supply

In the embryo, vessels from the cancellous portion of the vertebral body, as well as the ventral and the dorsal marginal vessels, run to the cartilaginous plate. These vessels do not penetrate the portion of the disk that later will become the nucleus pulposus or the annulus fibrosus. Hirsch and Schajowicz,[8] quoting Ubermuth, Boehmig and Schajowicz, said, "Thus, the nucleus pulposus and the annulus fibrosus are completely without vessels during every phase of the individual's life." However, they did note branches entering from the perichondrium into the calcified marginal epiphyses (seen when these growth centers begin to ossify at the age of 13), and the vessels disappear when the epiphyses fuse at about 20 years of age. They also noted a few vessels, but only in the outermost layers of the ligaments. Therefore, may it be that the blood supply from the central axial vessels, which runs only to the cartilaginous plate, and the dorsal and the ventral vessels carry toxins, infectious organisms or other noxious materials to interfere temporarily with the nutrition of the disk and its nucleus pulposus?

The bone adjacent to the disk is cancellous primarily; the disturbance usually occurs in children. Both factors are associated with good blood supply. May it not be, therefore, that this is among the probable factors to explain why the pathology is reversible and the calcification temporary?

Nerve Supply: Pain

Pain usually was a prominent symptom, except in the 7 cases in which calcification was noted in the disk as an incidental finding. The disk, however, is devoid of nerve supply, as shown by the studies of Hirsch and Schajowicz.[8] Fairly large bundles of nerve fibers have been observed outside the ventral and the dorsal ligaments, and thinner bundles accompanying the vessels could only be followed into the outermost parts of these ligaments. Nerves never have been observed to penetrate the ligaments to their junction with the disk, nor into the annulus.

An attempt will be made to explain the symptom of pain in the *disappearing* and in the later stages of the *dormant* form of disk calcification; also, the absence of this symptom in the earlier stage of the *dormant* and the *silent* forms.

It is suggested that there may be a correlation between certain facets of peritendinitis calcarea, an outstanding example of

TABLE 1 (*Continued*)

Case 12[20]	Silverman 1954	Female 13 months	—	—	—	—	Calcification, incidental observation	D-4, 11, 12, and L-1	Observed 9 years plus; D-4 and 12, disappearing at age 10 D-11 increasing L-1 unchanged	Silent
Case 13[20]	Silverman 1954	Female 2½ years	—	—	—	—	Calcification, incidental observation	D-2 and 7	Observed 4 years, no change	Silent
Case 14[20]	Silverman 1954	Male 6 months	+	—	+	—	Calcification, incidental observation. Recurrent upper respiratory infection. Hyperextension, muscle spasm	D-3 and 6	Observed 7 years, no change	Silent
Case 15[20]	Silverman 1954	Female 7 years	—	—	—	—	Calcification, incidental observation	D-3 and 6	More dense after 3½ years	Silent
Case 16[20]	Silverman 1954	Female 6 years	—	—	—	+—	Calcification, incidental observation, round back. Glomerular nephritis	D-3, 5, 8, 10, and L-1	1 study only	—
Case 17[20]	Silverman 1954	Male 7¾ years	100° F.	—	+	+	Torticollis, spasm	C-6	15 months	Disappearing
Case 18[20]	Silverman 1954	Male 11 years	—	—	+	—	Torticollis, right, spasm	C-5	Clearing 6 months	Disappearing

CASES 19 TO 21—AUTHOR'S CASES

Case 19	Rechtman, Hermel, Albert and Boreadis	Female 3 years	Normal	Normal	+	2 days	Torticollis, right cervical kyphos, muscle spasm	C-4	Partially disappeared, roentgen studies, October, 1951, and March, 1954	Disappearing
Case 20	Rechtman, Hermel, Albert and Boreadis	Male 33 years	?	—	3	Back, 2 weeks	Muscle spasm and dorsal kyphos in April, 1953. Calcification, incidental finding, 1949	D-7	1949—incidental observation. Practically disappeared between May, 1953, and March, 1954	Dormant
Case 21	Rechtman, Hermel, Albert and Boreadis	Male 5 years	102° F 2 weeks previously	Normal	+	See case history	Torticollis, muscle spasm	C-4	Practically disappeared in 21 months	Disappearing

TABLE 1

Case Reference	Author Year	Sex Age at Onset	Temperature	Sedimentation Rate (60 Min)	Pain Degree (1 to 4)	Duration of Pain	Clinical Findings	Location of Calcification	Duration of Calcification	Author's Classification
Case 1[1]	Baron 1924	Male 12 years	101.3° F.	32	4	4 weeks	Kyphos Tender D-12	D-12	1 year	Disappearing
Case 2[10]	Kohlmann 1931	Male 12 years	—	—	2	Several weeks	Tender upper dorsal	D-4	Almost gone 2 months	Disappearing
Case 3[13]	Lyon 1932	Male 8 years	High	—	2	12 days	Torticollis right	C-6	8 months	Disappearing
Case 4[8]	von Held 1934	? 10 years	Fever	—	3	2 months	Torticollis, left Hyperextension	C-2, 3 and 5	Almost gone 2½ months	Disappearing
Case 5[9]	Keyzer 1939	Male 2½ years	High	—	4	5 weeks	Stiff neck	C-2, 3 and 4	Unchanged, several months	—
Case 6[21]	Weens 1945	Female 5 years	100° F.	—	4	2 days	[Stiff neck, hyperextension, calcification, incidental finding 7 months before	C-6	1 year	Dormant
Case 7[14]	Roederer 1946	Female 10½ years	—	—	—	—	Dorsolumbar spasm, mild kyphos	D-10 and 11	Regression in size and density	Disappearing
Case 8[11]	Lasserre and Phelippot 1947	Male 11 years	—	—	—	—	Torticollis 1 month, spasm	C-6	—	—
Case 9[2]	Cohen et al. 1949	Female 6 years	—	—	Abdominal pain 3½ years, intermittent	—	Mild kyphos, spasm	D-10 and 11	Regression in size and density	Disappearing
Case 10[16]	Sandstrom 1951	Female 59 years	101° F.	25	4	20 days	Stiff, tender back; muscle spasm	L-1	About 8 months	Disappearing
Case 11[16]	Sandstrom 1951	Male 50 years	Normal	5	2	6 weeks	Negative	D-6, 7 and 8	About 6 months D-6 practically disappeared D-7 considerably reduced D-8 disappeared	Disappearing

and is discernible only on roentgen study. The dormant form initially had the characteristics of the silent and, subsequently, of the disappearing form. The relative depth of the lesion, both beneath the skin as well as from the periphery of the specific structure, the site of calcification, may modify the symptoms and the findings. At the shoulder it is known that when the calcific deposit breaks through its confines within the mesenchymatous tissue, where essentially there is no blood or nerve supply, and reaches tissues, that are rich in these elements, the resulting hyperemia and other tissue reactions may be responsible for pain and other symptoms, as well as for the absorption of the calcification frequently noted. The same process obtains in similar tissue elsewhere, and we are suggesting this mechanism in the disk.

The prime difference between idiopathic calcification in those areas in which it is more common and in the intervertebral disk is the age of occurrence. In 18 of the 21 cases herein recorded, 85.7 per cent, the calcification was noted between the ages of 6 months and 12 years, while calcific tendinitis usually occurs in adults. Another difference is the rarity of disk calcification, as reported in the literature. The authors, however, are inclined to agree with Neuhauser, who stated that while idiopathic "disc calcification may be unusual, it is not a surprising finding."

Differential diagnosis must be made between idiopathic intervertebral disk calcification and traumatic, inflammatory and other painful conditions of the spine and adjacent soft tissues. Because of the 3 cases reported in adults, the condition may need to be differentiated from the more usual degenerative type of disk calcification, as well as from ochronosis, which also is probably degenerative in nature and secondary to a metabolic disorder. A stiff neck in children is also an early sign of meningitis and poliomyelitis, and these also should be considered in differential diagnosis. It might be of interest to make roentgen studies of a group of children with acute onset of "stiff neck" to find if calcification of the disk is not more prevalent. If the results were positive, the etiologic factor might be more clear. Such roentgen studies were made on an occasional patient, but they were negative.

In all the reported cases recovery was complete, without recurrence or sequelae. The prime treatment is local rest, traction, immobilization and the use of analgesic drugs (salicylates). Sandström[15,16] and Sandström and Wahlgren[17] considered the disease to be a type of calcifying tendinitis, and Sandström used roentgen therapy in the treatment of his 2 cases. The first patient had pain for 20 days, the second for 6 weeks, after roentgen treatment was started. Our patient (Case 20) was of the opinion that he probably was not benefited by the roentgen therapy. Comparing the time before Sandström's patients were pain-free with that reported in the literature in others causes one to question the value of the modality used. However, if this treatment should be used as a therapeutic test in children, the dosage would, and should, be so small that if it did not help, there could be no question of possible harm to the growing centers of ossification.

SUMMARY

1. Three cases of idiopathic calcification, primarily in the nucleus pulposus of the intervertebral disk, are added to the 18 previously reported.

2. All idiopathic cases display a relatively similar and definite picture, except those observed incidentally, the *silent* form. Thus, they differ from cases showing permanent calcification of the disk, which usually is on a degenerative basis and occurs in adults.

3. Complete subjective recovery may be anticipated, usually in a matter of days, and in most instances the calcification of the disk disappears more gradually.

4. A morphologic explanation of the localization of calcification of the disk in children was suggested. It was also advanced concerning pain that is present in some patients and absent in others.

TABLE 2. FATE OF CALCIFICATION

1 Case	Case 8	No follow-up study
1 Case	Case 16	1 study only
	Time Not Stated	
2 Cases	Case 7	Regression in size and in density
	Case 9	Regression in size and in density
1 Case	Case 15	Increased after 3½ years
	No Change	
3 Cases	Case 5	Several months
	Case 13	4 years
	Case 14	Several years
	Multiple Involvement	
1 Case	Case 12	Disappeared in 2 disks between 9th and 10th years
		Increased in 1 disk
		Unchanged in 1 disk
	Dormant Disk Calcification	
2 Cases	Case 20	Incidental calcification, 1949
		Acute symptoms began April, 1953
		10 months later, practically disappeared
	Case 6	Incidental calcification
		7 months later acute symptoms began
		1 year later calcification *disappeared*
	Disappeared Between 6 Months and 2½ Years	
10 Cases	Case Numbers: 1, 2, 3, 4, 10, 11, 17, 18, 19 and 21	
21 Cases		

TABLE 3. DISKS INVOLVED*

SINGLE DISKS (13 CASES)		
7 Cervical	*5 Dorsal*	*1 Lumba[r]*
C-4 (2)	D- 2 (1)	L-1 (1)
C-5 (1)	D- 4 (1)	
C-6 (4)	D- 7 (1)	
	D-12 (2)	
2 DISKS (3 CASES) (ALL DORSAL)		
Contiguous Disks	*2 Normal Disks Between the Involved Disks*	*4 Normal Disks Between the Involved Disks*
D-10 & 11 (1)	D-3 & 6 (1)	D-2 & 7 (1)
3 DISKS INVOLVED (3 CASES)		
Contiguous Disks	*Almost Contiguous Disks*	
C-2, 3 & 4 (1)	C-2, 3 & 5 (1)	
D-6, 7 & 8 (1)		
4 DISKS INVOLVED (1 CASE)		
D-4, 11, 12 & L-1 (1)		
5 DISKS INVOLVED (1 CASE)		
D-3, 5, 8, 10 & L-1 (1)		

* The disk is designated numerically corresponding to the vertebral body just above it.

Note that the disk involvement is not generally at the site of junction of the relatively fixed with the more movable portions of the spine, as might perhaps be expected.

which is rotary cuff calcification of the shoulder, and idiopathic calcification in the disk. The tissues involved are similar in their mesenchymatous origin. Other similarities regarding etiology, physiology and pathology may also be tenable.

In the rotator cuff area of the shoulder, calcification may be due to irritation associated with factors that may be similar to those discussed in this presentation in relation to the intervertebral disk. Shoulder calcification is concerned frequently with the repeated minor traumata of daily function, in conjunction with static deformity or local poor body mechanics.

The signs and the symptoms that occur in idiopathic disk calcification may depend on the degree of irritation. In the disappearing form, among the symptoms noted were pain, tenderness to pressure, restriction of motion, deformity, rise in temperature, increased sedimentation rate, deformity and muscle spasm. Histologic studies of the sites of calcification, made by Sandström and Wahlgren,[17] showed alterative, proliferative and exudative changes, all the signs of inflammation. The silent form is asymptomatic, gives negative findings on physical examination,

21. Weens, H. S.: Calcification of the intervertebral discs in childhood, J. Pediat. 26: 178, 1945.

Calcification del Disco Intervertebral: Disparente, Dormiente e Silente

Summario in Interlingua

Tres casos de idiopathic calcification del disco intervertebral es addite al dece-octo previemente reportate in le litteratura. Le autores del presente articulo opina que tres typos de idiopathic depositos calcific pote esser distinguite. (1) In casos del typo "disparente" le patiente se plange de dolores, e le roentgenogramma monstra calcification intra le disco. Post plure septimanas o menses studios roentgenologic monstra que le calcification ha disparite. (2) In casos del typo "dormiente" depositos calcific es primo observate como roentgeno-constatation incidental. Subsequentemente il ha symptomas acute. Ancora plus tarde, studios roentgenologic revela un disparition progressive del depositos. (3) Le termino "silente" es applicate a un typo de casos in que le calcification del disco es etiam observate como roentgeno-constatation incidental sed in que nulle altere signos o symptomas es causate.

Le etiologia de calcificationes idiopathic non es definitemente cognite. Le autores del presente articulo opina que le factores etiologic es plus o minus identic in omne histos mesenchymal, con le exception del typo chronic describite per Schmorl. Isto ha un base degenerative.

Le calcification es un lesion local que pote formar se como reaction a agentes noxie que causa un local disordine nutritional e un disvitalisation histologic via un mechanismo biochimic. Un inadequate o arrestate circulation in le disco e le altere histos mesenchymal explica probabilemente le formation de un deposito calcific in le presentia de certe factores stimulante. Isto differe ab le reactiones avascular, inflammatori, e cicatricio-histic que occurre in altere formas de histos conjunctive sub simile conditiones.

Le autores presenta lor conceptiones per correlationar certe factos observate in peritendinitis calcaree e calcification in altere histos conjunctive (mesenchymal) con observationes facite in le genere idiopathic de calcification del disco hic presentate. Signos e symptomas pote depender del grado de irritation. Illos etiam pote esser absente usque le deposito calcific se approxima al peripheria o usque illo penetra in le histos adjacente que es frequentemente ric in apporto nervose e sanguinee.

Es presentate un breve discussion del diagnose differential e del therapia. Recuperation occurre in omne patientes.

5. The ages ranged from 6 months to 59 years; 18 of the cases were in children, 85.7 per cent.

6. The etiology of idiopathic calcification of the disk is unknown, because it has not been fully established exactly what causes lime deposits to form. The authors agree with Sandström[15] that noxious agents result in local nutritional disturbance with devitalization, and that in mesenchymatous tissue (disk) calcification occurs as a local lesion, perhaps as an aberrant reparative process. While many factors have been postulated as being of etiologic significance, it is our opinion that they constitute merely a stimulation, which may result in calcification or activate a previously silent calcification.

7. The subclassification of the idiopathic type of disk calcification into disappearing, dormant and silent forms is suggested.

8. Seven of the patients had torticollis, and the other 2 with involvement in the cervical region had *stiff neck*. Roentgen study of the cervical spine might be considered for children with wry neck, to note if calcification of the disk is present.

9. The subject is presented to stimulate interest, study and research in the hope of proving the etiology of idiopathic disk calcification and increasing our knowledge of the disk problem in general.

REFERENCES

1. Baron, A.: Über einen Neuerkrankung der Wirbelsaule, Jahrb. Kinderk. 104:367, 1924.
2. Cohen, R., Burnip, R., and Wagner, E.: Calcification of the intervertebral disc in a child, Ann. West. Med. & Surg. 3:202-204, 1949.
3. Friberg, Sten: Discussion with Dr. Friberg when he attended the 1953 Alumni Meeting of the Hospital for Joint Diseases, New York, N. Y.
4. Harwood, Mark: Personal communication, 1954.
5. Held, von, H. J.: Zur Frage der Zwischen- [illegible] ontgeno- Ztschr.
6. Hirsch, Carl: Personal communication, 1953.
7. Hirsch, Carl, Paulson, Sten, Sylven, Bengt, and Snellman, Olle: Biophysical mesenchymal tissues. VI. Characteristics of human nuclei pulposi during ageing. Acta orthop. scandinav. 22:175-183, 1953.
8. Hirsch, Carl, and Schajowicz, Fritz: Studies on structural changes in the lumbar annulus fibrosus, Acta, orthop. scandinav. 22:184-231, 1953.
9. Keyzer, J. L.: Intervertebral calcinosis, Maandschr. V. Kindergeneesk. 8:467-473, 1939.
10. Kohlmann, G.: Röntgendiagnostik der Wirbelsäule, Verhandl. deutsch. Roentgen-Gesellsch. 23:48, 1931.
11. Lasserre, C., and Phelippot, G.: Discite calcifiante intervertébrale, Rev. d'orthop 33:494-500, 1947.
12. Lyon, E.: Kolkablagerungen in der Zwischen-wirbelscheibe in Kindesalter, Ztschr. Kinderk. 53:570-574, 1932.
13. Neuhauser, E., Radiologist-in-Chief, Children's Medical Center, Boston: Personal communication, 1953.
14. Roederer, C.: Calcification de plusieurs disques intervertébraux chez une fillette, Arch. françaises de pédiatrie 8:285-287, 1946.
15. Sandström, C.: Calcification of the intervertebral discs and the relationship between various types of calcifications in the soft tissues of the body, Acta radiol. 36:217-233, 1951.
16. ———: Peritendinitis calcarea: a common disease of middle life: its diagnosis, pathology and treatment, Am J. Roentgenol. 40: 1, 1938.
17. Sandström, C., and Wahlgren, F.: Beitrag zur Kenntnis der Peritendinitis Calcarea (sog. Bursitis Calculosa) speziell vom [illegible] Gesichts-
18. [illegible] der Hinterflache der Wirbelbandscheiben, Fortschr. Geb. Röntgenstrahlen 40:629-634, 1929.
19. Schmorl, G., and Junghanns, H.: Archiv und Atlas der normalen und pathologischen Anatomie in typischen Röntgenbildern, die gesunde und kranke Wirbelsäule in Röntgenbild, Leipzig, Thieme, 1932.
20. Silverman, F. N.: Calcification of the intervertebral discs in childhood, Radiology 62:801-815, 1954.

minutes with or without heat lamp at about 65° to 94° C. (150-200° F.). With the gloves on, coat the outside of the mold with plastic, apply glass cloth 18 x 30 cm. (7″ x 12″), and smooth carefully but rapidly. The glass cloth is the same as that used for covering boats; it can be bought from Benshoff and Co., Gauge 20.

Cover it with more mixture, another layer of glass cloth, etc., until 4 layers of glass cloth have been smoothed on. Each layer of glass cloth is preceded and followed by a



FIG. 1. Two thicknesses of 6 in. Johnson & Johnson fast setting plaster of Paris 12 in. long have been wet and smoothed onto the patient's foot. It is now almost hard and ready to be removed. The foot is in a neutral position with the ankle dorsiflexed to 90°.

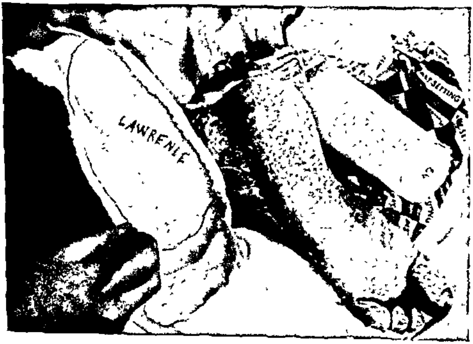


FIG. 2 The thin plaster mold has been removed, and is being labeled and marked for future cutting Care must be taken not to bend or to crease it.

22

Making Plastic Arch Supports*

A. A. KIRK, M.D., AND H. M. KUNKLE, M.D.†

With new materials available, we are finding easier and better methods of doing things. Our interest in arch supports stemmed from the fact that one of us could not find a pair that felt comfortable. The first support was made of glass cloth and plastic, with positive and negative molds. The same general construction is used, but the positive molds now have been eliminated, and the hard-setting plaster is included as part of the support. We realize that there are many ways of varying the procedure, but ours at present is as follows:

METHOD OF CONSTRUCTION

Place the patient on the abdomen with shoe and sock off. Bend the knee to 90° and dorsiflex the foot to 90°. Make sure that the foot is not turned in or out. Mark pressure points on patient's foot with ink (part of this comes off on plaster mold). Take 2 layers of 6 in. fast-setting, hard-set Johnson and Johnson plaster 30 cm. or 12 in. long. Ostic and Melmac should be satisfactory. Wet the plaster with warm water and place it on the foot. Smooth it down, particularly through the arch and round the heel and the edges, making sure that no air bubbles form. If some pin holes still remain in the plaster, they help the plastic to soak in. The patient usually can hold the legs and the feet in this position without too much difficulty, but a rest may be used. Allow the plaster to harden (about 5 to 15 minutes Fig. 1).

Loosen the mold round the edges. Have the patient work the toes and then the foot to help break the mold loose. Lifting it gently on the edges, remove it without bending. Moisten the areas that are marked with the ink stain. Press out on them to mold properly in order to prevent pressure points later. Mark on the inner sides for trimming, and write the patient's name on the inner surface with pen or pencil (Fig. 2). These marks remain, and can be seen on the finished product unless trimmed or scraped off (Fig. 3). Wrap with care, and warn the patient against denting or crushing.

The doctor's part usually ends here. We were fortunate in finding a man who was interested in the problem. After showing him how to make the supports, their construction was turned over to him. He uses the following procedure:

Dry the mold bone dry over a lamp or in an oven.

Put on rubber gloves.

Mix 4 parts (about 60 Gm. or 2 oz. per support) of Ortho Bond A with 1 part (15 Gm. or ½ oz.) Ortho Bond B, supplied by Vernon Benshoff and Co., Pittsburgh 30, Pa.

Mix thoroughly in a cup or a glass (the same glass can be used time after time until it fills up with residue). Use a teaspoon for mixing. Take a spoonful and rub into the inside of the mold, using the fingers.

Wash off the gloves and spoon with rubbing alcohol. Dry and powder the gloves, but do not remove them.

Turn the mold over on a sheet of paper and allow to dry and drain for 15 to 30

* Presented at the meeting of the Association of Bone and Joint Surgeons at Virginia Beach, Va., March, 1955.

† Portsmouth, Va.

minutes with or without heat lamp at about 65° to 94° C. (150-200° F.). With the gloves on, coat the outside of the mold with plastic, apply glass cloth 18 x 30 cm. (7″ x 12″), and smooth carefully but rapidly. The glass cloth is the same as that used for covering boats; it can be bought from Benshoff and Co., Gauge 20.

Cover it with more mixture, another layer of glass cloth, etc., until 4 layers of glass cloth have been smoothed on. Each layer of glass cloth is preceded and followed by a

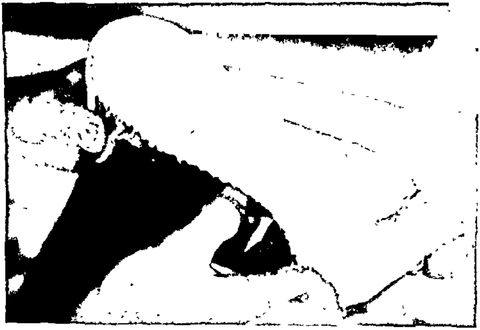


FIG. 1. Two thicknesses of 6 in. Johnson & Johnson fast setting plaster of Paris 12 in. long have been wet and smoothed onto the patient's foot. It is now almost hard and ready to be removed. The foot is in a neutral position with the ankle dorsiflexed to 90°.

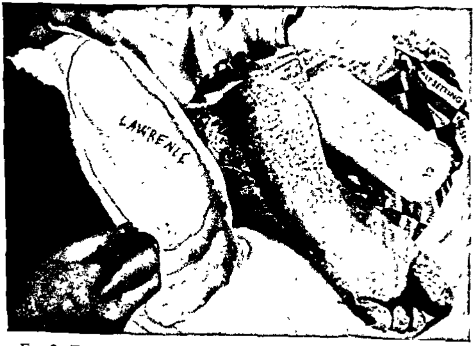


FIG. 2. The thin plaster mold has been removed, and is being labeled and marked for future cutting. Care must be taken not to bend or to crease it.

coat of mixture. In other words, after applying a layer of glass cloth, be sure to apply another layer of mixture before applying the next layer of glass cloth. Apply an extra coat of mixture on the inner side, wash your gloves and spoon, and again powder your gloves to keep them from sticking. More or fewer layers of glass cloth may be used, depending on the patient's weight. The above usually will take care of a patient weighing up to 200 lbs. if trimmed about 1 cm. or ½ in. up on each side. This prevents the foot from sliding laterally.

Allow to harden overnight, trim, sand or grind down the edges, fit to the patient, and trim off areas where it rubs bony prominences. (The front edge usually is cut about even with the metatarsal heads.)

Recheck in 1 week and sand down, trim or scrape off at pressure points. This is made easier with electrically driven rotary grinding tools. It should again be checked at the end of a month, or as often as is necessary. Metatarsal pads may be glued to the support, or they may be built up on the support out of plastic, but these usually are not necessary.

These arches usually hold up for 2 to 3 years or longer (Fig. 3). They have been found to be particularly useful in cases of painful heels, arch strain and calluses under metatarsal heads. They have been made mostly for men, but a few women have had them for severe foot trouble. Also, they have been suggested in cases of rheumatoid arthritis.

We believe that this is a good method, but the drawbacks are as follows:

DISADVANTAGES

The material is hard to scrape or sand This may be done with power tools.

The front edge wears into the shoe. After

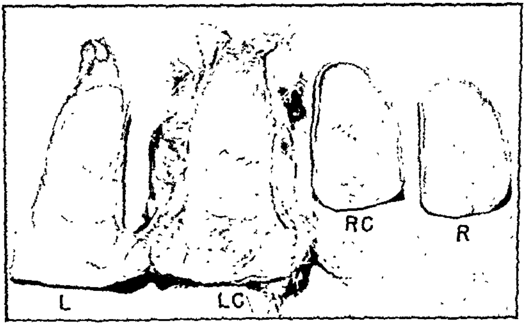


Fig. 3. *(L)* 1 coat of plastic has been applied to the inner surface of the mold. *(LC)* Layers of plastic and glass cloth have been applied to the plantar surface of the mold. It is now ready for cutting to size. The cloth has a tendency to separate from the mold at the heel. *(RC)* The support has been rough cut, usually with a jigsaw. The edges still need to be sanded. *(R)* This support has been worn for 2 years It has an extra layer of plastic and glass cloth on its inner surface. Those to the left do not have this extra coat.

about 6 months, it may develop a sharp edge that may cut the socks. This edge may be scraped with a knife or rounded off with sandpaper. The mixture hardens fairly rapidly. Your procedure should be carried out rapidly.

The glass cloth has a tendency to kick up round the heel (Fig. 3). Hold it down with your hand until it is firmly stuck, or tie it down with string.

If the mold is not completely dry, the plastic does not stick well. If, after completion, it shows a tendency to separate at the edges (Fig. 4), it may be repaired with a small amount of plastic rubbed into this area.

It requires a shoe that is a size larger, and it has a tendency to mark the side of the shoe after about 3 to 6 months. The sides may be sanded to minimize this.

It is hard and may rub bony prominences, making them sore if not sanded or scraped down in these areas.

It causes some slapping in the shoe when walking. This may be softened by a thin layer of sponge rubber or felt under the heel part of the support.

It may crack. If it does this, another layer of glass cloth and mixture may be added to the inside or the outside to strengthen it.

If it is made for a painful heel and is not drained properly, the plastic may puddle inside the heel and will not relieve the pressure on the plantar surface. It does not cure painful heels, but it makes the patient much more comfortable while they heal.

A special precaution in marking it for a painful heel is to be sure to have it high round the back so as to make a good cup, and thereby prevent the tissues from flattening out. This lessens weight-bearing on the sore spot.

If the plastic is not used within a year from the time it was opened, it may not harden properly. One set of supports was ruined because plastic was used that was 3 to 4 years old.

If heated too hot while drying, it may form bubbles. Do not heat it above boiling.

ADVANTAGES

The main advantages are that it is made to fit the individual foot. It may be added

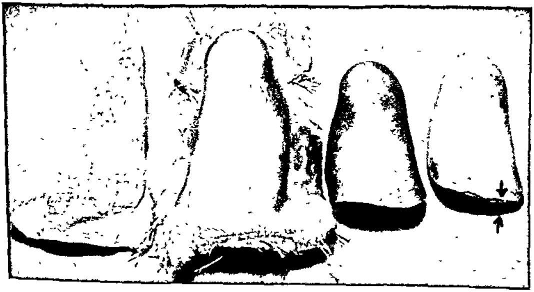


FIG. 4. On the plantar surfaces of the molds in Figure 3, as shown on the extreme right here, the front edge shows signs of wear and the tendency of the plastic to separate from the mold if the mold is not completely dry at the time the plastic is applied. This may be repaired by using a small amount of plastic locally.

to or reinforced at any time, plastic alone or plastic and glass cloth being used.

Pressure may be applied or relieved wherever needed.

It may be remolded after it has hardened when heated to about 100° C. or 212° F.

When making the mold, the plaster mold is included as part of the finished support. It has the other usual advantages of other removable arch supports.

COMMENTS

We have made from 30 to 40 supports in the past 3 years, with about 90 per cent good results. Many of these patients had tried other supports and pads without sufficient relief of pain. Most of the bad results were due to ordering them for patients with difficult or indefinite diagnoses, in the hope that the supports would help them. Some bad results were due to soreness from rubbing bony prominences, but these were corrected. The conventional metatarsal pads, longitudinal arch supports, Thomas heels and metatarsal bars are used, but the molded plastic arch supports are resorted to when sufficient improvement is not obtained.

MATERIALS NEEDED

20 wt. glass cloth
1 lb. Ortho Bond A
¼ lb. Ortho Bond B
Rubber gloves
Rubbing alcohol
Talcum powder for gloves
Newspaper
Rags
Spoon
Small drinking glass
Scissors
150 watt bulb and reflector
Magnetic jig or coping saw
Aviation shears
Emery stone
Sandpaper

ACKNOWLEDGMENT

The authors are indebted to Mr. Delbert W. Culpepper, for his invaluable assistance, and to Mr. James L. Ligon, for his photographs.

Preparation de Plastic Supportos del Volta Plantar

Summario in Interlingua

Plastic supportos del volta plantar es facite sin modulos positive e negative. Le modulo original deveni parte del supporto. Le patiente jace super le abdomine con genu e le articulation del pede flectite a 90 grados. Duo stratos de emplastro a solidification rapide e firme (6", Johnson & Johnson) es humectate e modulate super le pede. Post solidification le emplastro es removite e marcate al superficie interior pro le detondimento subsequente. Quando le emplastro es ben desiccate, quatro partes de Ortho Bond A e un parte de Ortho Bond B (Vernon Benshoff & Co , Pittsburgh 30) es miscite e applicate al superficie interior del emplastro modulate que es revertite pro drainage e solidification. Alora le superficie exterior es coperite con le mixtura, e un strato de panno vitree es applicate. Stratos additional del mixtura e del panno es applicate in alternation usque quatro stratos del panno es usate. Le sequente die, post solidification complete, le margines es detondite e polite con papiro-smerilio. Un septimana plus tarde le supporto es re-examinate e adjustate per medio de papiro-smerilio in sitos de prominentias ossee. Iste typo de supporto ha le avantages usual del supporto mobile. Illo se ha provate bonissime pro talones dolente. Il es possibile a non importa qual tempore adder nove stratos de panno pro allargar o reinfortiar le supporto. Su durabilitate es inter duo e sex annos. Nos lista le disavantages de iste typo de supporto e mentiona proceddimentos pro evitar los. In le caso de patientes de alte peso corporee on pote adder un strato al interior o al exterior del supporto.

23

Calcareous Tendinitis at the Elbow

ROBERT E. VAN DEMARK, M.D., F.A.C.S., AND
ARNOLD K. MYRABO, M.D., F.C.A.P.*

Calcareous tendinitis at the shoulder is a well-recognized entity, characterized by pain, local tenderness, muscle spasm and later atrophy in the shoulder showing a typical calcareous deposit in the musculotendinous cuff on roentgenographic examination.[1] At the elbow, the same condition occurs, but with much less frequency, and usually it is located in the common tendon of the extensor muscles attaching to the lateral epicondyle. Although it is no less disabling in the latter location, the correct diagnosis may not be established because of the rarity of occurrence at the elbow, and the condition is not given prompt and adequate treatment for relief of pain and loss of function. Anatomically, the condition at the elbow differs from that at the shoulder; the process is located in a thick, strong tendon which is less apt to rupture over the deposit and is not surrounded by a large bursa to aid in absorption should spontaneous rupture occur. The usual textbooks of orthopaedic surgery make little or no mention of this disabling entity. Hughes,[2] in a review of the subject in 1950, stated that he was able to find only 9 cases that had been reported.

At the elbow, with involvement of the common extensor tendon, the condition closely resembles *tennis elbow* with localized pain, tenderness and swelling in the region of the lateral epicondyle. There are roentgenographic findings just distal to the epicondyle of a calcific density of variable size and contour; in some cases it may resemble a loose body. With conservative treatment, the more chronic features of *tennis elbow* are said to persist.[2] Pathologic studies of the condition at the elbow are apparently very limited in number. Typical findings are well illustrated in the following:

CASE HISTORIES

Case 1. A white female, aged 25, was seen first on December 23, 1952, with a painful right elbow of 1 month's duration. There was no history of injury. The condition had been worsening gradually, and, following some ironing 2 days previously, the patient had been unable to use her arm. She had been examined roentgenographically by her family doctor, who found an apparent loose body over the lateral aspect of the right elbow, and she then had been referred for specialized treatment. The patient carried the arm in a sling.

Examination showed acute tenderness over the anterolateral aspect of the right elbow with localized swelling. The arm was most comfortable with the elbow in a neutral position. Motion in all directions was limited markedly because of pain, and could not be measured with consistency and accuracy. The roentgenograms which the patient brought with her showed a calcareous density laterally adjacent to the joint, the margins were well demarcated and strongly suggestive of a loose body at the margin of the joint (Fig. 1).

The patient was admitted to the hospital, and exploration of the area was performed. After incision of the skin, a calcareous tendinitis was found in the extensor tendon over the lateral condyle. This was incised, and, under pressure,

* Sioux Falls, South Dakota.



FIG. 1. Anteroposterior roentgenogram of elbow with a well-demarcated calcific deposit immediately adjacent to the joint that simulates a loose body.

a toothpastelike material exuded out through the incision. The cavity was curetted gently, and biopsy was made on the cavity wall; the skin and the subcutaneous tissue were closed with interrupted silk sutures, and a cast was applied. The relief from pain was immediate and dramatic.

The patient's postoperative course was uneventful, and she was dismissed from the hospital on December 24, 1952 She obtained a



FIG 2. Postoperative result in Case 1. Complete restoration of function and relief of pain.

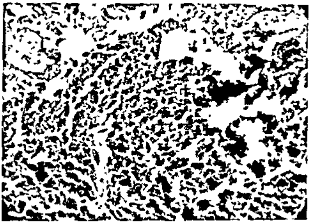


FIG. 3. Biopsy specimen from the tendon margin of the deposit in Case 1. Note the marked acellularity, small fragment of cartilage and ovoid bodies.

complete return of function with no residual pain or disability. Subsequent roentgenographic examination showed very little residual calcification (Fig. 2).

Pathologic examination was reported as follows:

"Gross Examination. There is a grumous, rather granular opaque mass of tissue, much like tooth paste aggregating an area measuring 16 x 11 x 9 mm. All tissue is rather friable, easily fragmented and uniform throughout.

"Microscopic Examination. The vast majority of the material is acellular, composed of granular basophilic debris Along one margin there are small fragments of cartilage showing well-preserved nuclei. These fragments are surrounded by similar basophilic debris. In a few areas there are rounded masses of varying sizes that are more eosinophilic, simulating degenerating tendon bundles Scattered throughout there are degenerating mononuclears and variable numbers of neutrophils. Some fibrin is observed.

"Diagnosis. Acute tendinitis with degeneration and calcification."

Case 2. A white female, aged 35, was seen on June 1, 1954, because of an acutely painful right elbow of 2 months' duration. There was no known injury, and the patient had been unable to use her arm for any of her usual housework. Previous physiotherapy treatments had been unsuccessful in relieving pain or restoring function.

Examination showed acute tenderness and swelling on the lateral aspect of the elbow. All motions were limited by pain, particularly in their extremes. Roentgenographic examination

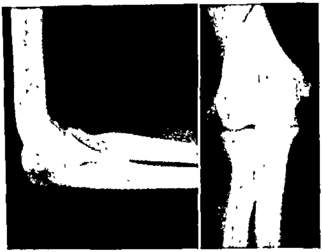


FIG. 4. (Case 2) Anteroposterior and lateral roentgenograms showing a calcific density lateral and distal to the lateral epicondyle of the humerus.



FIG. 6. Biopsy specimen from Case 2. In the upper portion of the section there is a relatively normal tendon. The lower portion shows extreme degeneration of the tendon fibers, with granularity and calcification.

showed a calcareous deposit in the soft tissues adjacent to the lateral epicondyle (Fig. 5).

The patient was admitted to the hospital and underwent surgery. The calcareous deposit in the extensor tendon was excised. It was of milk-like consistency, and spurted out of the tendon on incision. A portion of the wall was removed for biopsy after curettement of the cavity, the skin and the subcutaneous tissue were closed with interrupted silk sutures, and a cast was applied. The relief of pain was marked. The patient was dismissed from the hospital 3 days later.

The subsequent course was entirely normal, and at the end of 3 weeks the patient was completely asymptomatic and had a normal range of motion in the elbow.

Checkup roentgenograms, taken subsequent to surgery, were reported by the roentgenographic department as follows: "Subsequent film shows this calcification has apparently been removed." (Fig. 6.)

The pathologic report of the biopsy material was as follows:

"Gross Examination. There are 3 fibrous, opaque to grayish-yellow, irregular tissue fragments, the largest measuring 5 x 4 x 3 mm. Included separately is considerable granular, grayish-yellow debris, grossly suggestive of crystal formation.

"Microscopic Examination. Repeated sections are examined. In part, the tissue is tendinous in origin, dense eosinophilic, showing



FIG. 5. Postoperative result in Case 2.



FIG. 7. Biopsy specimen from Case 2. A zone of vascular granulation tissue separates the relatively normal tendon above from the lower degenerating tendon with its acellularity, granularity and dark areas of calcification.



FIG. 1. Anteroposterior roentgenogram of elbow with a well-demarcated calcific deposit immediately adjacent to the joint that simulates a loose body.

a toothpastelike material exuded out through the incision. The cavity was curetted gently, and biopsy was made on the cavity wall; the skin and the subcutaneous tissue were closed with interrupted silk sutures, and a cast was applied. The relief from pain was immediate and dramatic.

The patient's postoperative course was uneventful, and she was dismissed from the hospital on December 24, 1952. She obtained a complete return of function with no residual pain or disability. Subsequent roentgenographic examination showed very little residual calcification (Fig. 2).



FIG. 2. Postoperative result in Case 1. Complete restoration of function and relief of pain

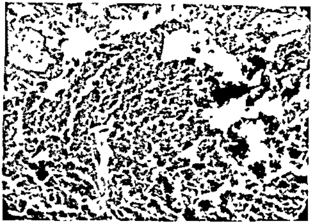


FIG. 3. Biopsy specimen from the tendon margin of the deposit in Case 1. Note the marked acellularity, small fragment of cartilage and ovoid bodies.

Pathologic examination was reported as follows:

"Gross Examination. There is a grumous, rather granular opaque mass of tissue, much like tooth paste aggregating an area measuring 16 x 11 x 9 mm. All tissue is rather friable, easily fragmented and uniform throughout.

"Microscopic Examination. The vast majority of the material is acellular, composed of granular basophilic debris. Along one margin there are small fragments of cartilage showing well-preserved nuclei. These fragments are surrounded by similar basophilic debris. In a few areas there are rounded masses of varying sizes that are more eosinophilic, simulating degenerating tendon bundles. Scattered throughout there are degenerating mononuclears and variable numbers of neutrophils. Some fibrin is observed.

"Diagnosis Acute tendinitis with degeneration and calcification "

Case 2. A white female, aged 35, was seen on June 1, 1954, because of an acutely painful right elbow of 2 months' duration. There was no known injury, and the patient had been unable to use her arm for any of her usual housework. Previous physiotherapy treatments had been unsuccessful in relieving pain or restoring function.

Examination showed acute tenderness and swelling on the lateral aspect of the elbow. All motions were limited by pain, particularly in their extremes. Roentgenographic examination

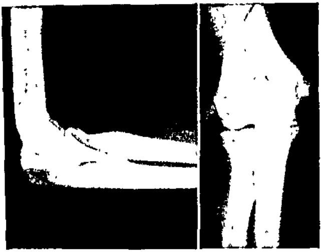


FIG. 4. (Case 2) Anteroposterior and lateral roentgenograms showing a calcific density lateral and distal to the lateral epicondyle of the humerus.



FIG. 6. Biopsy specimen from Case 2. In the upper portion of the section there is a relatively normal tendon. The lower portion shows extreme degeneration of the tendon fibers, with granularity and calcification.

showed a calcareous deposit in the soft tissues adjacent to the lateral epicondyle (Fig. 5).

The patient was admitted to the hospital and underwent surgery. The calcareous deposit in the extensor tendon was excised. It was of milklike consistency, and spurted out of the tendon on incision. A portion of the wall was removed for biopsy after curettement of the cavity, the skin and the subcutaneous tissue were closed with interrupted silk sutures, and a cast was applied. The relief of pain was marked. The patient was dismissed from the hospital 3 days later.

The subsequent course was entirely normal, and at the end of 3 weeks the patient was completely asymptomatic and had a normal range of motion in the elbow.



FIG. 5. Postoperative result in Case 2.

Checkup roentgenograms, taken subsequent to surgery, were reported by the roentgenographic department as follows: "Subsequent film shows this calcification has apparently been removed." (Fig. 6.)

The pathologic report of the biopsy material was as follows:

"Gross Examination. There are 3 fibrous, opaque to grayish-yellow, irregular tissue fragments, the largest measuring 5 x 4 x 3 mm. Included separately is considerable granular, grayish-yellow debris, grossly suggestive of crystal formation.

"Microscopic Examination. Repeated sections are examined. In part, the tissue is tendinous in origin, dense eosinophilic, showing

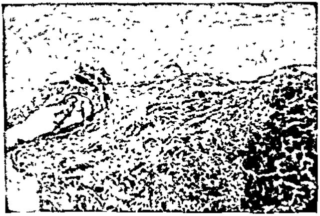


FIG. 7. Biopsy specimen from Case 2. A zone of vascular granulation tissue separates the relatively normal tendon above from the lower degenerating tendon with its acellularity, granularity and dark areas of calcification.

scattered spindled nuclei. There are extensive areas of degeneration with actual deposition of calcium salts. In these areas, tissue is more basophilic. I see no significant cellular infiltration or evidence of malignancy. Smears made of the aspirated material reveals much granular, calcareous debris without significant nucleated forms.

"Diagnosis. Degeneration of tendon with calcification."

DISCUSSION

The exact cause of this calcareous degeneration in younger individuals is not apparent. In neither case reported was there a history of trauma. Various methods of treatment have been advocated, including irradiation,[3] immobilization and surgery.[2] When the calcareous deposit is well localized and outlined, and of a large size, and the roentgenographic examination shows the deposit to be well demarcated from the surrounding tissues, incision and removal of the deposit are the treatment of choice, since it ensures thorough and rapid evacuation of the deposit (Figs. 2, 5), which otherwise may persist for a long period, particularly in the lesions of larger size which cannot be absorbed readily, as in Case 1. In each of the 2 cases, the deposit was under pressure, so that on incision it drained and decompressed spontaneously. The decompression probably is a factor in the dramatic relief of pain. When the calcific deposition is irregular and spotty, needling and aspiration of the area will aid in the resolution of the process and restoration of function.

REFERENCES

1. DePalma, A. F.: Surgery of the Shoulder, Philadelphia, Lippincott, 1950.
2. Hughes, E. S. R.: Acute deposition of calcium near the elbow, J. Bone & Joint Surg. 32-B:30-34, 1950.
3. Young, H. H.: Calcified bursitis, Proc. Staff Meet. Mayo Clin. 19:250-253, 1944

Tendinitis Calcari al Cubito

Summario in Interlingua

Es presentate duo casos de tendinitis calcari al cubito. Le patientes esseva juvene adultos sin historia de trauma. Le constatationes roentgenologic e pathologic es discutite. In ambe casos incision chirurgic con evacuation del deposito resultava in rapide alleviamento del dolor, restauration de functiones normal, e normal presentation roentgenographic. Quando le deposito calcari es ben localisate e demarcate, evacuation complete es effectuabile per incision e ablation. In altere casos, in que le deposito calcari es irregular e sporadic, punctionamento con aspiration es de adjuta in le resolution del processo e le restauration del functiones.

Section III

Motorist Injuries and Motorist Safety

CLINICAL ASPECTS

(Part 1)

GUEST EDITOR: JACOB KULOWSKI, M.D.
Saint Joseph, Missouri

1

General Introduction: Accident Prevention, Reduction of Injuries and Aids to Recovery

The high level of motorist injuries and the appreciation of the multiple causation of motorist accidents have instigated epidemiologic methods of study and control. However, as in any stage of transition, changes in ideas and in practices have not yet been crystallized, terminology is not well established, and the extent of new methods is not well known. It is the author's purpose to associate the ideas of three intersecting levels of approach to motorist injuries and safety—accident prevention, reduction of injuries and clinical aspects—in order that a measure of orientation and unity of action may be achieved.

Is it logical to link aids to recovery with supplementary and classic methods of motorist safety? It seems logical to combine diagnosis and treatment with etiology, pathology and prophylaxis of motorist injuries. It is not only logical but urgent in view of the possible adoption of the gas turbine for ground transport, which will forge even stronger connections between problems in aviation medicine and general medicine.

PRIMARY MOTORIST SAFETY

Motorist accidents derive from intrinsic or human factors and extrinsic or inhuman factors Motorist safety, therefore, is ensured by driver proficiency in conjunction with the safety performance of the vehicle and highway facility. One clinical deterrent to driver proficiency derives from poor physical condition, which causes dangerous driver behavior by interfering with functions essential to motorist safety, such as impaired vision. Several physical clues in this regard were noted by me in the more than 50 distinctive accessory diagnoses that had been made in autopsy studies of 29 motorist fatalities.* Physical, as well as psychological or mental, limitations stress the need for adequate standards of driver licensure and their maintenance, together with safer automotive design, both from the human-engineering and the crash-impact engineering points of view.

REDUCTION OF INJURIES

Crash-impact engineering, which governs this phase of motorist injuries and safety, may be described as the art of improving automotive design to reduce and/or moderate crash injuries and raise the level of survival during crash decelerations. The most important implications of this statement have to do with so-called survivable accidents, i.e., in which the occupant compart-

* These included cardiac hypertrophy, 4; previously amputated leg, 1; various degrees of atherosclerosis, 14; sclerosis of the mitral valve, 1; fatty liver, 1; anthracosis of the lungs, 5; cystadenoma of the pancreas, 1; retention cysts of the kidneys, 1; nephrosclerosis, 4; bilateral cataracts, 1; pleural adhesions, 3; chronic splenitis, 1; active rheumatic myocarditis with Aschoff bodies, 1; pulmonary fibrosis, 1; diverticulosis descending colon, 1; nodular hypertrophy of the prostate gland, 2; leiomyoma of the uterus, 1; hemartoma of the spleen, 1; focal fibrosis of the epicardium, 1; calcified nodule of the lung, 1; cholesterolosis of the gallbladder, 1; chronic duodenal ulcer, 1; recanalized old coronary thrombosis, 2; and accessory renal artery, 1.

2

Etiology of Motorist Injuries

The etiology of motorist injuries is rooted in physical laws governing inertia, deceleration and dissipation of kinetic energy that express interactions with the automotive environments.

EXTERNAL AUTOMOTIVE ENVIRONMENT (VEHICULAR)

To begin with, moving vehicles build up kinetic energy. Under normal conditions of deceleration or stopping, this kinetic energy is dissipated gradually and safely through heat resulting from braking and tire friction. The opposite obtains in a collision when the rapid and ungoverned dissipation of energy causes structural damage and deformation, and, frequently, injury and death of occupants. Mathematically expressed, the kinetic energy is arrived at from the product of the mass and the velocity squared divided by twice the force of gravity. The geometric relationship that speed (velocity) has to the resultant forces that are generated in motor-vehicle crashes causes high orders of energy that are best expressed in decelerative rates in gravities (g's) or multiples of weight.* Thus, when the speed is doubled the results are quadrupled. However, the mass or weight factors involved in the *equations* should not be overly subordinated to the speed

If nothing else happened in vehicular crashes except the damage to automotive property, the most impressive thing about a crash (assuming that no people were hurt is the fact that tremendous amounts of energy were absorbed through deformation and collapse of structure. However, except when anthropometric dummies replace them in experimental cars for crash purposes, people are often involved in accidents. By the same token, then, injuries represent manifestations of human bodily absorption of energy.

Acceleration and Deceleration

Like all fundamental physical phenomena, crash accelerations and decelerations are not easy to understand. One reason for this derives from the failure to differentiate clearly between applied and inertial forces. Another is the tendency to associate the very abrupt and brief forces in crashes with the relatively longer acting ones that occur in aircraft maneuvers and human centrifuge machines. It should be clearly understood that crash conditions are characterized by their

* Calculation of decelerative rates at 20 and 40 miles per hour (mph); assuming the vehicle weighs 4,000 pounds and the impact crushes it 1 foot (decelerative distance), which represents the actual stopping distance.

Deceleration equals $\frac{(\text{velocity})^2}{2x1}$ equals $\frac{(20x1.467\ [\text{ft./sec.}])^2}{2x1}$ equals $\frac{(29.3)^2}{2}$ equals $\frac{860.8}{2}$ equals 430.4 ft./sec.^{2}

Gravity equals 32.2 ft./sec. per sec., therefore $\frac{430.4}{32.2}$ equals 13.4 g's.

Same case for a 40 mph crash is: $(40x1.467)^2$ equals $\frac{(58.6)^2}{32.2}$ equals 53.4 g's.

Calculation of kinetic energy for the above case: K.E. equals ½ mv^2 equals ½ $\frac{w}{g}$ v^2 equals ½x $\frac{4,000}{32.2}$x58.6^2 equals 214,000 foot pounds. (Fig. 1).

brevity and large forces, and that human tolerances to the latter increase as the crash duration decreases. Further, the briefer the duration the less time or chance there is to overcome the inertia of the body fluids. However, under such conditions, the greatest crash-producing stresses are exerted upon the supporting tissues, as the ligaments, the bones and organ attachments. In other words, under longer acting accelerations and decelerations there are volumetric blood shifts, while, during the briefer accidental changes of velocity, the actual stress limits of the supporting tissues become the most important considerations.

Moreover, no matter how brief are the decelerative rates expressed in g's, it is instructive to remember that the specific gravity of both human and inhuman structures rises at the same rate as the decelerative forces exceed the gravitation of the earth. However, other physical characteristics of the human body naturally remain the same. Therefore, at three g's one weighs the same as a full-sized marble statue. Powerful shock-like forces of inertia easily cause structural deformations and collapse, also fractures and injuries of the soft parts, under such conditions.

Due to his sitting position in the crash vehicle, man's chief accelerative and decelerative forces are experienced in a transverse direction, i.e., at right angles to the body axis. Most frequently, these forces act chest to back; sometimes, from back to chest; and also from side to side Crash forces also may act in the long axis of the body: seat to head (positive); or head to seat (negative). Examples of these various directions of force are impaction of the steering controls by the driver's chest, rear-end collision effects, side impacts, vertical dislocations in which the head strikes the car roof, forcible ejection and landing on the ground with the head, the body or on the buttocks; to which may be added centrifugal forces from spins and roll-overs; and tumbling action from end-over-end roll-overs. It is evident that crash deceleration may be experienced in any body orientation, but it is experienced ideally in the transverse direction.

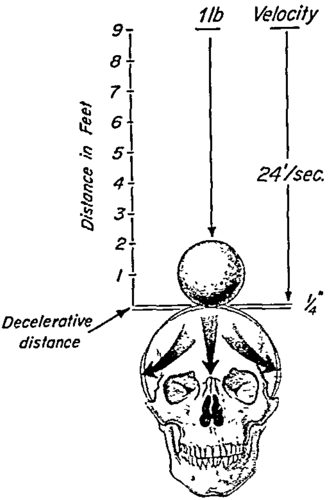


FIG 1. Illustrating the forces that the skull is able to resist: a 1 lb. weight falling 9 ft. reaches a velocity of 24 ft. per second and acquires 288 foot-second units of energy (or 86 lbs. to the sq. in. of skull); assuming this deforms the skull ¼ in. The forces and the pressures developed in this case simulate what happens when the skull is decelerated otherwise through this same distance from impacting undamaged portions of a crash-decelerated vehicle. (After Souttar)

INTERNAL AUTOMOTIVE ENVIRONMENT: OCCUPANT

Nothing happens to occupants of moving vehicles until their velocity relative to their immediate environment is changed. When the latter occurs, occupants first are accelerated and then decelerated on impact. The

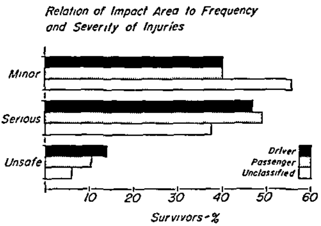


FIG. 2. From the standpoint of severity of injuries received, it makes little difference where one sits during a crash, according to the author's findings in this regard in a series of over 500 motorist survivors. In larger series the back seat is 3 times safer than the front one.

kinetics of the human body under crash conditions may be best understood when each side of the equation, force equals injury, is considered separately. In regard to the force conditions alone, those on the inside of the vehicle differ chiefly from those on the outside in the extremely short decelerative distances that the human body undergoes, as compared with relatively longer ones resulting from the crumpling of the vehicular structure. In other words, a human head

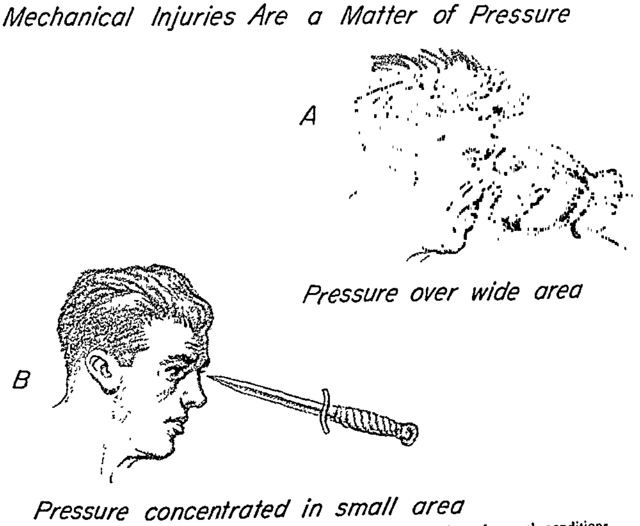


FIG 3 The smaller the area of pressure that structure exerts under crash conditions and/or upsets, the more penetrating the resultant injury will be. Also, the converse is true Other qualifying factors have been mentioned in the discussion, such as the direction of force, the duration of its action (impulsive or crushing), the magnitude, the portion of the body affected and the general physical condition of the rider.

striking a hard surface is brought to a stop in a fraction of an inch (Fig. 1). On the other hand, the undamaged portion of the vehicle may do the same thing in a matter of several feet. In this way, a 15 to 20 g crash on the outside is magnified to several hundreds, or even thousands, of g's on the inside in regard to the human occupant. The actual jolt the occupant experiences depends on his bodily contact with the undamaged portions of the vehicle in relation to general structural peaking. If he makes contact before peaking (maximum deceleration), then he rides out the rest of the decelerative run on undamaged structure and benefits from a roll-with-the-punch effect (also afforded by safety restraining gear). In other words, his decelerative distance is being increased thereby. The converse is true when he makes contact after the structure has reached its maximum deceleration, since the occupant's deceleration distance becomes shortened proportionately, and damage to tissues is thereby increased.

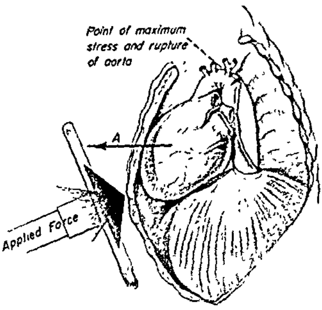


FIG. 4. A classic example of initial applied force against chest by steering controls and the resultant reactive or inertial forces exerted chiefly at the superior attachment of the aorta, which frequently causes a rupture of the aorta at this point. See further discussion in chapter on injuries of the chest and the abdomen.

A more subjective sense of what happens under crash conditions may be appreciated from the fact that a 25-pound ball weighs 1,000 pounds when it is traveling at 25 mph and decelerated within an inch.

The force conditions are similar to those already mentioned in regard to the external environment; they involve the physics and the mathematics of human mass, velocity and decelerative distances on impact, and how these relate to the progressive or irregular (peaking) crumpling of structure. Stapp's anthropometric dummy, when it was decelerated abruptly at speeds of flight ripped away two lap straps, went through 1-in. thick board windscreen and then hurtled straight ahead for another 700 feet before coming to a stop.

The mechanical variables encountered under crash force conditions may be likened to and visualized by the various reactions of bowling pins to the bowling ball.

From the standpoint of force effects on tissues, several predisposing factors are said to be operative. These include direction of impact, which may be governed by preimpact position of the occupant and his relation to potentially dangerous surfaces (Fig. 2). The body area (chest and head) affected often determines the general effects, as well as the local damage. The surface area over which the force is applied (impulsive or quick or crushing and prolonged) determines the type of injury. Mechanical pressure over larger areas of the body produce contusions or bruises, while pressure over smaller areas lead to penetrations (Fig. 3) or lacerations. The limits of elasticity of the different tissues, as well as the general condition of the motorist at the time of accident, also are important factors in the production of morbidity and/or mortality. Finally, the effects of inertial forces are sometimes far more damaging than the externally applied forces, and they are more difficult to evaluate (Fig. 4). Briefly stated, survival depends upon the time-pressure effects versus tolerances of the vital parts

Cornell Aeronautical Laboratory, Inc.: Head impact research, Final Report, January, 1948.

DeHaven, Hugh: Mechanical analysis of survival in falls from heights of 50—150 feet, War Med. 2:586, 1942.

Dye, E. R.: Automobile Crash Safety Research Report, No. YB-846-D-1, Liberty Mutual Insurance Co., Dec. 31, 1953.

———: Kinematic Behavior of the Human Body During Crash Deceleration, Jan. 4, 1950.

Einstein, A., and Infeld, L.: The Evolution of Modern Physics, New York, Simon & Schuster, 1950.

Evans, F. G., *et al.:* The role of tensile stress in the mechanism of femoral fractures, J. Bone & Joint Surg. 33A:485, 1951.

———: Strength of human compact bone (engineering techniques), Am. J. Surg. 83: 326, 1951.

Gallie, W. E., and LeMesurier, A. B.: The transplantation of fibrous tissue in the repair of anatomical defects, Brit. J. Surg., vol. 289, 1924-1925.

German Aviation Medicine, World War II, vol. 1, Department of the Air Force, 1950 (out of print).

Glasser, Otto: Aviation medicine and Elasticity of tissues *in* Medical Physics, vol. 2, Chicago, Yr. Bk. Pub., 1950.

Goldman, D: Mechanical forces acting on aviation personnel, J. Aviation Med. 17:426, 1946.

Gratz, C. M.: Tensile strength and elasticity tests on human fascia lata, J. Bone & Joint Surg 13:334-340, 1931.

Gurdjian, E. S., *et al.:* Studies on skull fractures with special references to engineering factors, Am J. Surg. 78:736, 1949.

Haber, Henry: The Physical Environment of the Flyer, Randolph Field, Texas, Air University, United States Air Force, School of Aviation Medicine, 1954.

Haboush, E. J.: Photelastic stress and strain analysis in cervical fractures of the femur, Bull. Hosp Joint Dis, vol. 13, 1952.

Hartog, J., and Den, P: Mechanics, New York, McGraw-Hill, 1948.

Hass, G M Relations between force, major injuries and aircraft structure with suggestions for safety design in aircraft, J. Aviation Med. 15:395, 1944.

Higdon, A., *et al* Engineering Mechanics, New York, Prentice-Hall, 1949.

Jannsen, Murk: On Bone Formation, Its Relation to Tension and Pressure, London, Longmans, 1920.

Lemon, H. B., and Ference, M.: Analytical Experimental Physics, Chicago, Univ. Chicago Press, 1943.

McMaster, P. E.: Tendon and muscle rupture, J. Bone & Joint Surg. 15:3, 1933.

Martin, E. G., *et al.:* Muscular strength and symmetry, Am. J. Physiol. 47:29, 1918.

Mathewson, J. H., and Severy, D. M.: Automobile Impact Research, 42nd National Safety Congress, Oct. 21, 1954.

Moore, Mark B.: Principles of Experimental Stress Analysis, New York, Prentice-Hall, 1954.

Moritz, A. R: Mechanisms of head injury, Ann. Surg. 117:512, 1943.

Popov, E. P.: Mechanics of Materials, New York, Prentice-Hall, 1952.

Reichenbach, Hans: The Rise of Scientific Philosophy, Berkeley, Univ. California Press, 1951.

Roth, Herman: Physical Factors Involved in Head-on Collisions of Automobiles, Highway Research Board Proceedings, Dec., 1951.

Senery, D. M., and Mathewson, J. H.: Automobile Barrier Impacts, Bull. 91, Highway Research Board Proceedings, 1954.

Severy, D. M.: Automobile Crash Effects, California's Governor's Safety Conference, Oct. 7, 1954.

Souttar, H. S.: Physics and the Surgeon, Springfield, Thomas, 1948.

Stapp, J. P.: Effects of mechanical forces on living tissues. 1. Abrupt deceleration and windblast. J. Aviation Med. 26:268, 1955.

———: Human Exposure to Linear Deceleration, Air Force Technical Report No. 5955, Dec., 1951.

———: Human tolerance to deceleration, summary of 166 runs, J. Aviation Med. 22: 42, 1951.

Steindler, Arthur: Kineseology, Springfield, Ill., Thomas, 1955

Sutherland, G. A.. The physics of blast, Lancet 2:641, 1940.

White, A. J: Engineering pilot study to determine injury potential of steering assembly designs, Motor Vehicle Research, Project No. 8041, June, 1955.

Woodson, W E: Human Engineering Guide for Equipment Designers, Berkeley Univ. California Press, 1954.

3

Pathology of Motorist Injuries*

The bodies in this series of fatalities consisted of 23 males and 6 females, whose ages ranged between 3 and 78 years. All intervening decades were represented, the third one most frequently. Sixteen had been drivers, 15 of them men. One had sat in the right front seat, 3 in the back seat, and 10 could not be classified according to seating.

PRIMARY INJURIES

Primary injuries were classified into 3 chief categories: topical or surface injuries (contusions, abrasions and lacerations); fractures; and internal injuries. In 29 motorist fatalities, the incidence of the first two subgroups was established. There were 111 surface lesions—45 contusions, 35 lacerations and 31 abrasions—chiefly on the exposed parts of the body. The frequency rates of fractures was: chest, 69 per cent; extremity, 50 per cent; head, 32 per cent; pelvis, 32 per cent; and face, 14 per cent

It is becoming increasingly better known that fractures often are too severe a criterion of the results of crash forces, because not infrequently serious internal injuries occur without concurrent overlying skeletal injury. In this small series, for example, 7 persons or 25 per cent of the injuries to the brain were not associated with skull fractures. Moreover, a similar number of people with intrathoracic injuries did not have fractures of the chest wall. Even more significant was the fact that several of these brain and pulmonary lesions resembled so-called blast injuries. Of course, the converse also was true, there being 3 fractures of the chest wall without evidences of intrathoracic injury.

The vital dividing line between morbidity or clinical pathology and mortality is made up of the internal injuries (Table 1), the common denominator in all of these fatalities. The combinations of body cavities affected included: head, chest and abdomen, 6; head and chest, 5; head and abdomen, 3; chest and abdomen, 4; head, chest and peripheral, 1; chest, diaphragm and abdomen, 1; chest and diaphragm, 1; and chest and peripheral, 1. Approximately 75 per cent of primary internal injuries occurred above and below the diaphragm. Contusive and lacerative injuries were about evenly divided in these areas. Brain lesions were predominantly contusive, with a tendency to be concentrated in the area of the pituitary body. The frequent relationship of cerebral contusion to cerebral concussion is obvious.

The critical area of the body in regard to motorist casualties has shifted from the head to the chest, due to the increasing number of crushing injuries here and the fact that pulmonary ventilation outranks all other vital functions of the body. Contusive lesions of the lungs were twice as frequent as lacerations of the lungs. However, the distinction between them is not too clear cut here because of the spongy character of these organs. There were 17 crushing injuries to the

* Records from which the present data were extracted were obtained from 3 sources: 12 from the University of Kansas Medical Center (Dr. Robert E. Stowell, Director and Head of the Department of Pathology and Oncology); 15 from the Missouri Methodist and 2 from the St. Joseph Hospitals (Doctors Marvin W. Morse and William J. Hunt, Pathologists).

TABLE 1. FREQUENCY OF PRIMARY INTERNAL INJURIES IN 29 MOTORIST DEATHS

	PATHOLOGIC ANATOMY	CONTUSION	LACERATION	TOTAL NO.
Intracranial	Brain	15	3	18
	Optic nerve	0	1	1
Above the diaphragm	Lungs	7	3	10
	Pleura	0	3	3
	Mediastinal*	7	6	13
Diaphragmatic		0	2	2
Below the diaphragm	Gastro-intestinal	4	0	4
	Spleen	3	2	5
Intraperitoneal	Liver	1	3	4
	Omentum	1	1	2
Retroperitoneal	Kidney	2	2	4
	Adrenal gland	1	1	2
Pelvic	Urinary bladder	2	1	3
	Anorectal	0	1	1
Peripheral	Vascular	1	1	2
	Nervous	0	1	1
		44	31	75

*Includes 4 pericardial lacerations, 2 lacerations of the heart and 7 contusions of the heart in 10 subjects.

TABLE 2. SUMMARY OF PRIMARY MEDIASTINAL INJURIES IN 10 MOTORIST DEATHS

AGE	SEX	STATUS	IMPACT	PATHOLOGIC ANATOMY	SURVIVED
65	M	Driver	Forward	Laceration left pericardium	7 days
74	M	Driver	?	Contusion left ventricle	9 hours
55	M	Driver	Rear end	Contusion right atrium, laceration anterior pericardium	3 hours
36	F	?	?	Laceration anterior pericardium	7 days
28	M	?	?	Contusion left ventricle	4 hours
68	M	Driver	Forward	Rupture right pulmonary artery, laceration right atrium, posterior tear left pericardium	Dead at scene of accident
29	M	?	?	Laceration right atrium, contusion left atrium, contusion left ventricle	8 days
11	F	Rear seat	Ejected and run over	Contusion left ventricle	8 hours
25	M	?	?	Contusion left ventricle	3 days
27	F	Driver	Lost control	Contusion left ventricle	6 days

chest, 14 of which were very severe (8 gerontols).

Ten subjects revealed injuries to the heart, the pericardium and the great vessels (Table 2), and probably were due to a combination of applied and inertial forces. Pathologists have known these lesions for some years. The clinical recognition still is lagging far behind in this regard. These lesions of the mediastinal structures were the primary causes of death in 2 cases, decisive causes of death in 1 case; contributory in 5 and non-

contributory in 2 cases of this series. The first 2 cases mentioned focus attention upon the tremendous shocklike forces that are required to cause immediate and massive irreversible circulatory failure. This kind of death epitomizes the so-called primary shock, and may occur without additional internal or external wound formations. There were 4 injuries to the pericardium and 10 injuries to the heart and the great vessels. The majority of the lesions of the heart were contusive; therefore, to a large extent they were reversible lesions. Oddly enough, severe crushing injuries of the chest were present concurrently in only 3 of these with mediastinal injuries. But all of them were associated with other concurrent serious lesions.

Two cases with mediastinal injuries were known to have had cardiac disease prior to accident. Issues were raised in regard to the causes of death in them. In both instances, autopsies were ordered by coroners. In each case postmortem studies showed that the causes of death were due indisputably to the gross injuries that had been received in the accident. In addition, it was found that old coronary disease had been present previously, but that these old lesions had become recanalized some time before the accident.

Injuries of the diaphragm are closely related to those involving the spaces above and below it and to the lower ribs as well. In this series, most of the lacerative injuries to the lungs resulted from penetration by broken ends of ribs. A similar mechanism of diaphragmatic injury occurred in 1 of the 2 injuries to the diaphragm in this series. This occurred in a 58-year-old male driver who died 3 days after admission to the hos-

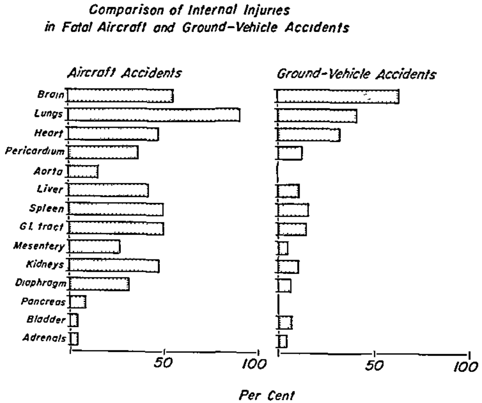


FIG. 6. The fatal aircraft injuries were reported and tabulated by Hass, whose basis was 30 necropsies. The ground-vehicle accidents (on the right) were tabulated by the author on the basis of 29 necropsies. Aortic and pancreatic lesions were conspicuously absent in the latter series; the larger percentages (except for the head) of lesions in aircraft accidents were notable.

pital. All his ribs on the left side were broken, also the left wrist. Postmortem examination revealed that the 10th rib had punctured the diaphragm and the pleura. About 500 cc. of blood was found in the peritoneal cavity, due to multiple rupture of the spleen, double fracture lacerations of the left kidney, and contusion and hemorrhage of the omentum. Above the diaphragm there were found bilateral atelectasis of the lungs, pulmonary congestion and edema, left hemothorax and slight terminal pneumonia. The bronchi were filled with sanguinopurulent exudate. Undoubtedly, these latter effects resulted, at least in part, from the dysfunction of the diaphragm. However, it is emphasized that intrathoracic injury and/or dysfunction is always associated with increased secretory activity of the bronchi and tendency toward pulmonary edema. Tracheotomy and/or aspiration, therefore, is frequently lifesaving.

The second case of diaphragmatic injury occurred in a 68-year-old male driver who was involved in a collision with a truck and died at the scene of the accident. The forces involved in this case must have been very great and localized to this region, because there were no injuries elsewhere in the body. The injuries received consisted of bilateral crushing injury to the chest (more on the left side and involving the sternum); gross bilateral tearing at the base of the diaphragm; and gross injuries to the heart, the pericardium and the great vessels, which were complicated by bilateral hemothorax.

Undoubtedly, the nature of these injuries (Case 2) can be correlated with a severe forward impaction on the steering controls. However, it should be remembered that gross injuries to the diaphragm may result from severe forces applied to either side of the body, and more subtly by vertically directed inertial forces (so-called positive or negative accelerations) On this basis (negative g), intrathoracic injuries may accrue from pressures of the liver and the spleen being transmitted through the diaphragm. The converse, or traction injuries of the mediastinum, may occur, but it is more characteristic of airplane accidents in small angle crashes or ditchings (positive g). Be that as it may, auto-crash and plane-crash fatal injuries are very similar (Fig. 6).

It should be noted that injuries below the diaphragm lend themselves to a more exacting anatomic classification: intraperitoneal and extraperitoneal (retroperitoneal and pelvic). Organs like the liver and the spleen, in the immediate vicinity of the diaphragm, were associated almost invariably with fractures of the lower ribs. It has been stated further that it requires more force to injure the liver than the spleen; therefore, in the presence of injury to the liver, one always suspects and looks for other evidences of intra-abdominal injury, especially that of the spleen. Occasionally, both liver and spleen will be found to be injured in the same individual. In this series there was 1 complete rupture of the liver, the rest being lesser lacerations, with 1 occurring in the subcapsular areas only and classifiable also as a contusive lesion.

Actually, there were 4 lacerative lesions of the liver, but 1 did not involve the capsule, and it is listed under contusive injuries for that reason. This injury was found in a 28-year-old male who died 4 hours after admission to the hospital with multiple injuries. All 4 cases with gross liver damage had fractures of the ribs and multiple other injuries as well. Liver injuries occurred in 4 men aged 28, 27, 29 and 55 years, 2 of whom were known to have been driving. As has been mentioned, 1 of these cases was associated with a ruptured spleen, another with a ruptured adrenal gland.

Three lacerative (rupture) injuries were noted in regard to the spleen: in a 3-year-old child who had been ejected, and in 2 male drivers aged 29 and 58 years. All were associated with fractures of the ribs. All had multiple other injuries as well. In 1 case there was a concurrent injury of the liver, with survival up to 8 days after admission to the hospital. The other 2 cases lived 30 hours

and 3 days after admission to the hospital. The patient who lived for 3 days also received injuries of the kidney and the omentum.

Injuries of the gastro-intestinal tract were relatively benign in portions that are freely mobile; less so in portions that are attached more firmly, such as the mesentery. The most severe lesion was found in a 64-year-old male who had been found partially pinned underneath his overturned pick-up truck and died about an hour after admission to the hospital. He was admitted in severe shock, due to large blood loss into both pleural cavities and the peritoneal cavity. Both sides of his chest were crushed. There was noted, in addition, multiple tears of the terminal ilium and mesentery, with herniation of the contused cecum through the injured mesentery.

Three other contusive lesions were present; these involved the gastro-intestinal tract in 3 men aged 25, 30 and 58 years, all of whom probably were drivers. In 1, there was involvement of the omentum alone; in the second, the stomach and the intestines were affected, and in the third, the stomach, the esophagus and the duodenum were involved. Two of these 3 cases were associated with other lesions below the diaphragm—contusion of the spleen and the adrenal gland in one and ruptures of the kidney and the spleen in the other.

The retroperitoneal injuries involved the adrenal glands and the kidneys. Curiously enough, the kidneys were damaged on the right side of the body, while the adrenal glands were damaged on the left There were no associated injuries of the lumbar spine in any of these cases, as might have been expected from the force and the anatomic standpoint All these lesions occurred in young men. Associated injuries of the genito-urinary tract was observed in only 1 case—a rupture of the urinary bladder in a man aged 28 years who had been ejected forcibly from his car and died 14 hours after admission from severe and scattered internal injuries. There was no concurrent fracture of the pubic or other bones of the pelvis. This emphasizes again the fact that serious internal injuries do occur without overlying skeletal damage, and vice versa. This one and only lacerative lesion of the urinary bladder was thought to have been produced purely by increased hydrostatic pressure of a more or less full bladder at the time of the accident.

The 2 injuries of the adrenal glands were of special interest in view of the relationship of these glands to the maintenance of blood pressure, to shock, and to other stress reactions of the body. One of these patients died within 4 hours after admission, the other one in 3 days. The first death was associated with a huge retroperitoneal hemorrhage arising from the adrenal gland itself.

The 3 peripheral injuries refer to a severe extrathoracic hemorrhage due to a lacerated right subclavian artery and vein (direct impact of steering controls); a partially lacerated brachial plexus in the same case; and a reflex segmental arterial spasm (iliac or femoral) associated with a comminuted fracture of the ilium on the same side in another subject.

DIRECT COMPLICATIONS

The most important complication of motorist injuries is hemorrhage. Table 3 establishes the frequency of this complication in the different areas of the body already discussed in conjunction with primary injuries. From the standpoint of primary injuries, attention was focused upon the chest, the most important single region of injury, which is being made ever more notable by high-speed motorist accidents. However, considerations of the direct, immediate and early complications present a paradox. From this standpoint, it is to be noted that the intracranial area regains its classic rank, because here, above all other areas (except in the pericardial sac), disastrous consequences always follow upon space-occupying lesions or bleeding. These large accumu-

TABLE 3. SECONDARY COMPLICATIONS IN 29 MOTORIST FATALITIES

Pathologic Anatomy	Direct Complications	No.	Indirect Complications	No.
Intracranial	Hemorrhage	27	Fat embolism	3
	Cerebral edema	3	Meningo-encephalitis	2
	Cerebral softening	3		
Intrathoracic				
Pulmonary	Hemorrhage	9	Pneumonia	5
	Edema	9	Emphysema	2
	Atelectasis	7	Tracheobronchial exudates	10
	Congestion	13	Fat embolism	4
Pleural	Hemothorax	16	Hydrothorax	2
	Pneumothorax	4		
Mediastinal	Hemopericardium	3	Acute dilatation of the heart	1
	Mediastinal hemorrhage	4		
Diaphragm	Hemothorax	1		
	Hemoperitoneum	1		
Below the Diaphragm				
Intraperitoneal	Hemoperitoneum	8	Fat embolism kidneys	1
	Cecal hernia	1	Lower nephron nephrosis	3
	Hemorrhage, organs and viscera	5	Urinary cystitis	2
			Degenerated adrenal glands	2
Extraperitoneal	Retroperitoneal hemorrhage	5	Congestive organs	12
Total		119		49
Peripheral				
Subclavian artery	Massive extrathoracic hemorrhage	1		
Iliac artery			Reflex segmental arterial spasm	1
Brachial plexus	Paralysis arm	1		

Peripheral complications not mentioned above included massive extrathoracic hemorrhage, paralysis of the arm and ischemia of the lower extremity.

Other hemorrhages of the central nervous system included: petechial, 5; subarachnoid, 8, into the ventricles, 3; internal capsule, 1; and into the spinal canal of unknown origin, 1.

lations of blood (outside the head) recall to mind the huge gravity and antigravity collections of pus that derive from tuberculous and pyogenic osteomyelitis of the spine and the pelvis. However, it is the cardiorespiratory systems that are knocked out when he intracranial pressure gets too high! Of the rather numerous hemorrhages listed in Table 3, 10 were considered to be space-occupying ones, 9 of which were subdural and 1 a classic epidural hemorrhage. The latter occurred in a man of 25 years who died 3 days after hospital admission

Regarding the interpretation and the evaluation of hemorrhages found at autopsy, above and below the diaphragm, it should be remembered that some bleeding may continue after death. However, the massive amounts of blood noted in the pleural cavities, the abdominal cavity and especially in the retroperitoneal lumbar gutters can hardly fail to impress the clinician with the importance of replacement of blood loss in any case of primary shock. Such bleeding sometimes occurs in a retrograde fashion from fractures of the pelvis, and occasionally it ac-

cumulates from such small sources as the adrenal injuries.

INDIRECT COMPLICATIONS

The later complications of motorist injuries, both degenerative and infectious in origin are quite well known for the most part. Others, like fat embolism and lower nephron nephrosis, often are lost sight of during the exigencies of the earlier posttraumatic stages of these cases. One also is impressed from Table 3 by the rapidity with which congestive, pneumonic and tracheobronchial impediments to survival develop. The last also recalls the small limits of factors of safety provided in regard to adequate pulmonary ventilation; that is, the small airway at the glottis as against the 5 lobes of the lungs. Anatomic corollaries of a similar situation are to be found in the limited coronary circulation and the vulnerable vessels of the brain and their far-reaching results when injured.

CONCLUSIONS

A studied appraisal of primary injuries and direct and indirect complications that occurred in this series of motorist casualties brings into sharp relief the factors of shock in its broadest sense. There seems to be a tendency on the part of the body to recapitulate shocklike states during the convalescent stages after injury. This was recognized most clearly by Moon, who promulgated definite principles of primary and secondary shock from the standpoint of clinical pathology, which is supported by this study. Moon stated that early death from traumatic shock results from circulatory failure. Death, after 48 hours, often is the result of a combination of circulatory deficiency with renal deficiency and terminal pneumonia. Thus, secondary shock results from a combination of causes rather than, as usually has been supposed to be the case, from recurrent circulatory failure alone. Secondary shock results from the delayed effects of tissue injury, hemorrhage, infection, surgery and even unfavorable reaction to transfusion.

Clearly, then, we are becoming more sensitive to contributory causes of death in trauma. In order to further this objective approach to traumatic morbidity as well as mortality, more basic information with regard to etiology and the exact nature of tissue injury is needed. The fundamental concept is that it is the exception to have the total body damage as a result of the primary injury alone. Most likely this is due to interference with vital functions or loss of blood, the former of which is not even recognized as or conceded to be a wound in itself. From this standpoint, certain clinical implications emerge from the morbidity and mortality of motorist casualties that definitely and naturally insinuate themselves into the currently expanding fabric of accident and injury prevention in general, and motorists in particular.

BIBLIOGRAPHY

Gerber, S. R.: Analysis of fatal accidents in Guyahoga County for a period of three years, Ohio M. J. 37:17-20, 1941.

Grant, R. T., and Reeve, E. B.: Observations *on the General Effects of Injury in Man*, with Special Reference to Wound Shock, London, His Majesty's Stat. Off., 1951.

Halter, Bert L.: Non-penetrating trauma to the heart, Am. J. Surg. 90:237, 1955.

Hass, G. M.: Types of internal injuries of personnel involved in aircraft accidents, J. Aviation Med. 15:77, 1944

———: Relations between force, major injuries and aircraft structure with suggestions for safety in design of aircraft, J. Aviation Med. 15:395, 1944.

Hodges, H. H., *et al.:* Myocardial contusion, fatal, Ann. Int. Med. 34:248, 1951.

Kulowski, Jacob: Clinical Implications of Motorist Fatalities, Read Before the National Association of Coroners, Kansas City, Mo., Aug., 1955.

Moon, V. H.: Pathology of secondary shock, Am. J. Path. 24:235, 1948.

———: Shock; mechanism and pathology, Arch. Path. 24:642, 1937.

Moritz, Alan R., *et al.:* Cardiac contusion, Arch. Path. 25:445, 1938.

———: The Pathology of Trauma, Philadelphia, Lea & Febiger, 1954.

Rice, W. G., *et al.:* Acute hypertension and delayed rupture of the aorta, J.A.M.A. 147: 915, 1951.

Roetig, L. C., *et al.:* Traumatic rupture of the spleen, Am. J. Surg. 2:292, 1943.

Rushmer, Robert, *et al.:* Internal injuries produced by abrupt deceleration of experimental animals, J. Aviation Med. 17:511, 1946.

Strassman, G.: Traumatic rupture of the aorta, Am. Heart J. 33:508, 1947.

Wright, L. T., *et al.:* Traumatic rupture of the liver without penetrating wounds, Arch. Surg. 54:613, 1947.

4

Implications of Time Intervals Between Injury and Death

(Mortality Differentials)

On the basis of the 29 motorist casualty fatalities discussed in Chapter 3, there were 3 distinctive subgroups according to the interval of time between accidental injury and death, i.e., immediate, intermediate and delayed. The implications relative to these categories will be discussed in this chapter. By *immediate fatality* will be meant death at the scene of accident that followed upon the impact; by *intermediate death*, a time interval between injury and death up to 48 hours afterward; by *delayed mortality*, all deaths after 48 hours while these people still were in the hospital and from causes directly related to the particular accident (Fig. 7). In this series 3 persons died immediately; 19, within 48 hours after admission to the hospital, and 7, up to 8 days after being admitted to hospital. The rates of these different mortality subgroups were 10, 66 and 24 per cent, respectively.*

Considerations of these mortality time differentials implicate automotive design (Fig. 8), first aid, emergency care, and definitive diagnosis and treatment. Immediate fatalities represent a complete breakdown of human and inhuman protection under crash conditions. Of the 3 immediate deaths, 2 were gerontols and 1 a young man of 26 years. All 3 were involved in high speed forward collisions, and were drivers. The old men received serious chest injuries; the young one sustained severe pelvic injuries. Moreover, all 3 presented accessory findings indicative of lowered general physical condition at the time of the crashes. These deaths are human appeals for adequate medical standards of driver licensure and reasonable speed laws. Here, indeed, is a field for unified action by the two A.M.A.s *

Of the intermediate deaths, 10 people died a few hours after their arrival at the hospital, and 9 died later on. The average age of this subgroup of 10 people was 54 years, and contained 5 gerontols. The latter showed 4 intracranial, 7 intrathoracic and 3 abdominal injuries and 5 massive hemorrhages to account for death. Primary shock still was the obvious cause of death in this number. The average age of those who lived a little longer was 26 years, with 1 gerontol. Conditions contributing to the latter group deaths included 9 intracranial, 6 intrathoracic and 3 abdominal injuries and 3 massive hemorrhages. Already, however, the more indirect (secondary) complications were operating against survival. The rate at which these complications make their bid for the patient's life is amazingly fast, a fact that cannot be overemphasized. The most significant of these included: hyperpyrexia, 2; sec-

* John Moore, of Cornell Crash (Automotive) Injury Research, stated that 15 per cent of motorist casualties studied by them were killed outright (personal communication).

* American Medical Association and Automobile Manufacturers' Association.

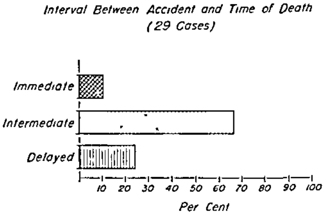


FIG. 7. The fact that the vast majority of deaths occur after the victims have been admitted to the hospital points up the therapeutic challenge of motoring injuries to both hospital and professional groups.

ondary shocklike state, 3; fat embolism, 1; lower nephron nephrosis, 1; and—fantastically enough—pneumonia, 2.

Up to this point it is evident that chest injuries and their complications are tending definitely to outrank all other types of injuries in regard to motorist deaths. This fact also stresses certain indications for emergency care, as well as definitive treatment later on: that is, facilities for replacements of large amounts of blood loss by blood and maintenance of vascular volume by plasma expanders; cardiac massage; and tracheotomy and bronchial aspiration for pulmonary ventilation. These are the minimal requirements for adequate emergency care of the dangerously injured persons in the outpatient department itself, unless immediate arrangements are at hand to cope with these emergency situations elsewhere in the hospital. The minor injuries, usually treated in outpatient departments of hospitals, cannot be neglected, nor their standards of care lowered; but the time has come when these same departments must be ready and set up for expanded lifesaving measures as well.

In this connection, naturally, even such grave responsibilities will have to be undertaken at times by the intern and the resident staffs. It behooves the medical school authorities to furnish medical students with more practical training along these lines before graduation, since there is little time between graduation and internship and the neophyte is already faced with the grim realities of motorist casualties. Furthermore, more attention to mechanical injuries in the medical schools will engender a greater interest in these most common conditions of man. Finally, most of these injuries occur at night, when young and vigorous interns and residents are better equipped physically to cope with the preliminary care of motorist victims.

Regarding the delayed fatalities, the average age was 38 years, and included 1 gerontol. Survival periods ranged from 3 to 8 days in this category. Conditions contributing to their deaths included: 7 intrathoracic, 4 intracranial and 2 abdominal injuries and complications, and 2 massive hemorrhages. The more indirect complications included meningo-encephalitis, 2; fat embolism, 3; pneumonia, 3; and lower nephron nephrosis, 3. These complications stress the fact that

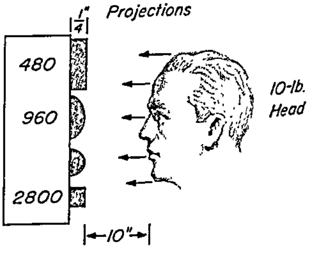


FIG. 8. Illustrating the effects of faulty automotive safety design (not to be confused with automotive safety engineering) in regard to increasing pressure areas from various projecting structures on the dash in causing facial (and other) injuries on impact. The smaller surfaces offer greater resultant forces. (After DeHaven)

motorist injuries are as much medical as surgical conditions. As a matter of fact, with increasing time after injury and little or no signs of improvement, this kind of patient actually is a medical rather than a surgical case. More medical participation in the treatment of motorist injuries would constitute a more realistic approach to the problem of motorist casualty survival.

It comes as a shock to realize that traffic victims—even those with obvious fractures of the extremities—are being transported without benefit of even the most primitive caliber of dressings or splints. With all that has been said and taught about first aid by both medical and nonmedical interests, this abysmal fact presents yet another of the paradoxes that abound in regard to the problems of motorist safety and motorist survival. However, this is remediable when the causes of this seemingly unhumanitarian attitude are analyzed. The indifference of laymen to the need for adequate first aid may be traced to 2 fundamental factors:

The first is the current fatalistic attitude of the public in regard to severe injuries of all kinds—probably engendered by the atom bomb and the apparent hopelessness of the injuries resulting from it. This attitude also hampers progress in civil defense plans.

The second involves the too complex methods of first aid that continue to be taught. Two examples should suffice to make this point clear. We expect lay groups to render first aid; but at the same time we overcaution them about moving injured individuals. Consequently, these victims literally are left to lie in their "road beds" until ambulance attendants extricate them. Also, while the so-called fixed traction method afforded by the Thomas type of apparatus is an excellent one, it is far too complicated for effective and rapid use by laymen, unless they have had specialized training and practice in its use.

Can these 2 cogent faults be eliminated? Yes.

First, aid in the field should be revised in accordance with primitive requirements (Fig. 9); that is, clean cover for wounds and minimum support for fractured extremities. Traction is not essential at this stage, when fast and safe transport to hospital usually is available. I have suggested a universal kind of telescoping light weight splint for this purpose



Fig. 9. Doctors and others will use simple types of splints, as demonstrated above, with regard to fracture of the ankle (*top*), fracture of the elbow (*center*) and compound fracture of the leg (*bottom*). The exigencies of accidental motorist injuries demand speedy, simple methods of splinting.



Fig. 10. More refined types of hospital splinting. (*Top*) Shanz type of knee bandage. (*Center*) Modified "Tobruk" plaster splint for fracture of the femur. (*Bottom*) Traditional and excellent Thomas splint for lower extremity fractures.

and a single package type of dressing. Undoubtedly, there are and will be other suggestions even more practical than my own. This will take serious thought all along the line by medical men.

Second, it is suggested that indoctrination in simpler methods should be concentrated upon 3 major classes of people: truck drivers; bus drivers; ambulance drivers and attendants. Police personnel ought to be relieved of this duty in the face of their other arduous ones related to various aspects of law enforcement and administration of traffic conditions. The others are usually just as available as the police anyway. First-aid material in commercial carriers would serve another purpose—that of being a constant reminder of safety practices and a chance to live up to what they stress—the safety practices of commercial carriers. A fair percentage of vehicular accidents involve commercial carriers of one kind or another.

Third, modern revisions of first aid ought to be undertaken by the medical profession and those allied to it, but it should be administered by the Red Cross and Civil Defense. In this way, perhaps, a full scale program of first aid can be built up, not only for motorist victims—whose need outranks all others now—but possibly for victims of even more catastrophic disasters to come.

What has been said is not intended to underestimate what has been done in the way of first aid. However, like all other problems of motorist safety and motorist injuries as a whole, this facet also needs to be unified by those most capable of developing the program. Doctors will have to give these less academic but no less important aspects of their work more serious consideration. The tremendous amount of thought and work that this entails can be cut down materially by activation at the local community level. For the medical profession, this means at the level of the county medical society.

To press the point further, an expansion of first aid will aid interns and residents immeasurably in easing the burdens of emergency care. For this reason, hospitals ought to take a more active part in the teaching of first aid. It goes almost without mention that the same humanitarian principles of first aid are to be maintained after the patient reaches the hospital (Fig. 10). There is of necessity much movement of accident cases within the hospital, especially in regard to roentgenographic examinations. The need for simplified methods of splinting (and trac-

tion) is perhaps as great there as in the field. Are hospital authorities cognizant of this and are they making every effort to improve the conditions?

Obviously, definitive treatment of motorist victims should be a continuation of alertness to and awareness of the major factors that produce primary and secondary shock.

More specifically, this calls actually for changes in therapeutic policies with regard to therapeutic priorities in the seriously and dangerously injured people. Basically, these relate to maintenance of the circulation, adequate pulmonary ventilation and maintenance of arterial volume, all of which may mean the same thing from the physiologic standpoint, but must be distinguished from one another from the standpoint of management.

Two ideas need to be clarified along these lines. First, that an individual in shock is not in a state of suspended animation but always is getting either worse or better. This is a dynamic concept, not a static one. This means that frequently there is little time for some of the niceties of accurate diagnosis and treatment. However, in the dangerously injured persons, the diagnosis ought not to be hampered by overconcern about the patient's chances in withstanding certain movements in the hospital, as roentgenographic and other laboratory methods. Those chances must be taken on the basis of the patient's individual requirements. There is no rule of thumb here; nor should there be. These risks, however, like those of anesthesia and surgery, which already have been minimized by medical progress, likewise will be minimized by progress in the more deft and considerate handling of these people.

SUMMARY AND CONCLUSIONS

Three major mortality differentials, with regard to time after injury when death took place, implicate and involve considerations of the human and the inhuman ingredients of motorist safety and motorist injuries. Immediate deaths from impacts and delayed deaths form about 25 per cent of the total mortality; which leaves approximately 65 per cent in the intermediate fatality rate. This area has focused chief attention upon first aid and emergency care, but much remains to be done in other areas of the over-all problems of motorist safety and motorist injuries.

Currently, the situation with regard to medical standards of driver licensure and improved crashworthiness of vehicles is most urgent. Not enough is being done in either classification. First aid and emergency care need to be made a more integrated program with definitive treatment and to be brought up to the latter's advanced standards of practice. More careful considerations of therapeutic priorities for the dangerously injured people have been suggested.

There is every reason to believe that the percentage rate of survivors will step up with improved automotive safety design, unless continued increasingly high engine power and speed cancel out these efforts. Put another way, it may be expected that the immediate mortality will be cut down proportionately as the art and science of crash-impact design advance. In all probability, these additional survivors will be in the dangerously injured subgroup—those attended by a high mortality. Therefore, it is emphasized that, in so far as medicine is concerned, its responsibilities will be increased from the standpoint of diagnostic and therapeutic challenges. It is further emphasized that first aid and emergency care will be increased proportionately. Moreover, during the period of transition to better design, there may even be an increase of injury-producing accidents due to foolish experimentation by foolish drivers—just to see what such design and safety gear will do in a protective way. In all of this there is a real challenge to the smaller communities of America to meet existing threats and conditions by means of hospital expansion and improvement in available services.

Practitioners will do well to realize that the problems presented by motorist casualties are clinical rather than either medical or surgical. Therefore, the seriously and dangerously injured people require a multifaceted therapeutic approach. The greatest single need in this respect—now—is more medical (internist) participation in any team which undertakes the responsibility of caring for motoring casualties.

BIBLIOGRAPHY

Hagman, Frank E.: Management of the wounded soldier *in* Surgery of Trauma, Philadelphia, Lippincott, 1953.

Hampton, Oscar, Jr.: Priorities and life saving measures in multiple injuries, South. M. J. 47:147, 1954.

Kulowski, Jacob: Emergency care of and ambulance organization for crash victims (and catastrophe) (symposium), Canad. Services M. J. 11:575, 1955.

Rowbottom, G. F.: Acute head injuries, Canad. Services M. J. 11:575, 1955.

5

Pilot Study: The General Morbidity

Data for this chapter were extracted from 661 motorist casualties* who had been injured severely enough to require inpatient hospital treatment and ultimately survived the effects of the crashes in which they were involved. The youngest was 3 months; the oldest, 82 years. All intervening decades were well represented: the third one, 23 per cent of the total; the ninth one, 1 per cent. Men and boys predominated in the first 4 decades; females were more frequent after that. Forty-six per cent were drivers (75% male); 29 per cent were in the right front seat (75% female); 8 per cent sat in the back seat (60% female); and the rest could not thus be classified, but they were occupants.

CLINICAL PATHOLOGY

A pattern of injury was sought from the angles of seating and the frequency rates of injury of the different parts of the body in accordance with topical, fracture and internal injuries. The first reference to seating in this series clearly established the fact that the driver got the worst of it, while passengers were less vulnerable, especially those who occupied the back seat. But from the standpoint of degree of *morbidity*, it was found—on the basis of 3 distinctive categories of morbidity (45% mild to moderate, 45% moderate to severe, and 10% dangerous injuries)—that the same rates of these injuries were received in all parts of the crash vehicle. From this standpoint it makes little or no difference where one sits under crash conditions.

Table 4 gives the relationship between seating and the nature of the injuries received, data having been extracted from 400 motorist accidents. In 294 accidents, only 1 person was injured per crash, while in 106 accidents, 2 or more individuals were injured per crash. The majority of the casualties in the single injury accidents were drivers. On the other hand, as was to be expected, when 2 or more people were hurt in 1 crash vehicle, there were more guest passenger victims than drivers. In the latter category, it is to be noted that a greater percentage of these had been sitting in the back seat, which should remove some of the complacency about this area being the safest seat in the car. The evaluation is a relative one only. The variations, between driver and passenger morbidity, mentioned above are eliminated naturally in large statistical evaluations with regard to mortality. In the long run, as many drivers are killed, for example, as any other kind of occupant. The reverse used to be stated.

Table 5 establishes the pattern of the overall injuries or criteria of the different parts of the body. These areas were chosen for medical purposes of diagnosis and management, but they may also be of value to engineers and research workers. Sixty-seven per cent

* This series of cases were admitted to the Missouri Methodist Hospital from late in 1949 until the end of 1954. They were treated by various members (the author included) of the medical, the surgical and the dental staffs. During this time, approximately 400 or more similar cases also were admitted and treated at the St. Joseph Hospital. They, however, were not included in this study, since the author thought that the records of 1 institution only would serve to lend greater validity to the statistical analysis.

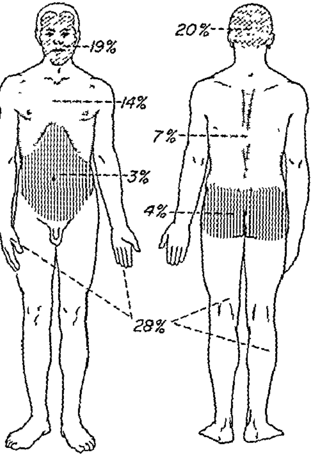


FIG. 11. Graphic representation of rates of general bodily involvement in 661 motorist casualty survivors according to different areas of the body.

TABLE 4. RELATION OF TYPE OF INJURY TO SEATING IN 400 MOTORIST ACCIDENTS

SEATING	%	PERCENTAGE AND TYPE OF INJURIES		
		EXTERNAL	INTERNAL	SKELETAL
A*				
Driver	68	43	18	39
Front seat, right	27	44	10	46
Back seat	5	50	0	50
B†				
Driver	38	48	13	39
Front seat	47	49	19	32
Back seat	15	50	19	31

* 294 accidents, 1 person injured per crash

† 106 accidents, 2 or more persons injured per crash

Note: Internal injuries include cerebral concussion. Skeletal injuries include fractures, dislocations, subluxations and severe sprains

TABLE 5. FREQUENCY OF INJURIES TO DIFFERENT BODY AREAS IN 661 MOTORIST-CASUALTY SURVIVORS (OVER-ALL INJURIES)

BODY AREA INJURED	NO. OF AREAS	%
Extremity	378	28
Head	273	20
Face	259	19
Chest	181	14
Trunk	97	7
Neck	62	5
Pelvis	52	4
Abdomen	35	3
	1,337	100

of the injuries involved the more peripheral or smaller body masses; 80 per cent affected the head and the face; 14 per cent affected the chest. On the other hand, the trunk, the abdomen and the pelvis combined made up only 19 per cent of the total number of injuries. From this standpoint the baseline ranking order of incidence was: extremity, head, face, chest, trunk, neck, pelvis and abdomen. Since the direction of impact is largely a function of the seated position, directional inertia from impact and body size, this pattern is of significance from the force-injury relationships (Fig. 11).

Table 6 lends further support to the hypothesis that an injury pattern exists for motorist casualties, although there is a slight change in the order of frequency with which topical injuries affected the same body areas analyzed in Table 1 (interchange between neck and abdomen). About 55 per cent received contusive and abrasive lesions; approximately 50 per cent suffered lacerations. Thirteen per cent of the contusions and abrasions affected 2 or more parts of the body (in the series as a whole, 80% of the cases had multiple lesions, approximately 1,300 injuries in all). Forty-eight per cent of the topical injuries affected the head and the face. Surface injuries were about 3 times more frequent than fractures. A sparing action of the soft parts that protect the under-

TABLE 6. FREQUENCY OF SURFACE INJURIES ON THE DIFFERENT PARTS OF THE BODY IN 661 MOTORIST-CASUALTY SURVIVORS

BODY AREA AFFECTED	NO. OF INJURIES	%
Extremities	315	32
Head	244	25
Face	224	23
Chest	99	10
Trunk	42	4
Abdomen	32	3
Pelvis	18	2
Neck	13	1
	987	100

lying tissues from harm by absorbing the energy of impact first is suggested in these figures.

Table 7 reveals a definite change in the injury pattern from the standpoint of fractures of the parts of the body studied in the previous tables.

The most striking drop in rank was that of the head. The rank increase of the pelvis was less thought provoking, since it still remained in the lower echelons. Eighty-nine per cent of the fractures involved the extremities, the face and the chest (Fig. 12). The upper and the lower extremities were affected about equally. Detailed discussions will follow in appropriate chapters.

The third medical link between crash force and its effects resided in the internal injuries (Table 8). The great preponderance of head injuries is due to the inclusion here of cerebral concussion. Without these injuries, the head, the chest and the abdomen would rank about the same. About 20 per cent of the entire series manifested some degree of cerebral concussion. Other internal injuries included: contusion and laceration of the brain, 2; contusion and laceration of the lungs, 3; ruptured gallbladder, 1; ruptured spleen, 2; ruptured kidney, 2; mesenteric hemorrhage, 1; and several others that were suspected but were not proved adequately on their charts for them to be in-

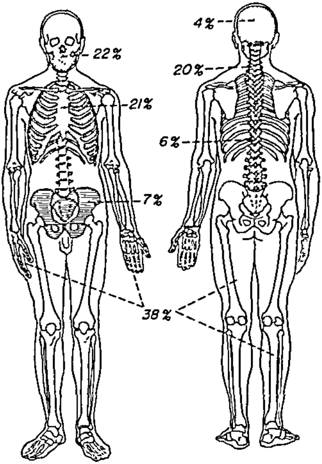


FIG. 12. Graphic representation of distribution of fractures in 661 motorist casualty survivors.

cluded here. However, such symptoms as tarry stools and incomplete bowel obstructions in several cases supplied strong presumptive evidence of contusive lesions of the gastro-intestinal tract. Complications, as

TABLE 7. FREQUENCY OF FRACTURES IN THE DIFFERENT PARTS OF THE BODY IN 661 MOTORIST-CASUALTY SURVIVORS

PART OF BODY AFFECTED	PERCENTAGE OF FRACTURES
Extremity	38
Face	22
Chest	21
Pelvis	7
Trunk	6
Head	4
Neck	2
	100

TABLE 8. FREQUENCY OF INTERNAL INJURIES IN 661 MOTORIST-CASUALTY SURVIVORS

BODY CAVITY AFFECTED	%
Head	80
Chest	10
Abdomen	10
	100

distinguished from primary injuries, were not given the prominence on medical records of survivors accorded them in autopsy records. Consequently, it is more difficult to evaluate these factors in survivors. Suffice it to say that complications included hemorrhage—the chief cause of shock—hydrothorax and pneumothorax, hemothorax, pulmonary edema, atelectasis and a small diaphragmatic hernia.

PRINCIPAL IMPACTS

Another approach to the establishment of injury-pattern predictability is shown in Table 9. It indicates the relationships between the principal impacts and the nature of the principal crash impacts. This table is based upon 328 motorist-casualty survivors, regardless of age, sex, seating or the number of people involved in the various accidents, or the degree of morbidity. However, there can be little doubt that the trends shown in this table give active support to the crash-impact point of view, which implicates source of energy as one of the most potent factors in the production of injury under crash conditions, regardless of the type of principal impact or impacts involved.

Viewed from another angle, the fact that 2 or more drivers were involved in the vast majority of these accidents would implicate poor driver practices rather than unreliable vehicular performance. This also is supported by the paucity of mechanical failures.

Finally, closer scrutiny of the tabulations also give some idea of the importance of forcible ejection and the magnitude of the forces that are generated by the different types of impacts. The function of linear (forward and rear) and angular impacts may be inferred to some degree from the effects. The smaller the angle, the smaller the forces that are generated, and vice versa. The function of speed is obvious in this respect. It is harder to evaluate the centrifugal forces involved in roll-overs and spins, not to mention tumbling actions of the vehicles (end over end).

CONCLUSIONS

A study of the clinical pathology in 661 motorist-casualty survivors indicates that a definite and predictable pattern of crash and/or upset condition injuries occurs. The most important implications of this statement have to do with diagnosis and treatment. For example, the frequency rates of inci-

TABLE 9. *RELATION BETWEEN* PRINCIPAL IMPACTS AND TYPE OF INJURIES IN 328 MOTORIST-CASUALTY SURVIVORS

PRINCIPAL IMPACTS	%	PERCENTAGE AND TYPE OF INJURIES EXTERNAL	INTERNAL	SKELETAL
Vehicular collisions*	26	50	14	36
Head-on vehicular collisions	15	40	20	40
Forcibly ejected from vehicle	13	51	21	28
Roll-over	12	48	15	37
Ran off road	8	45	15	40
Collision with fixed object	8	41	10	49
Collision from the side	5	54	6	40
Rear-end collisions	3	41	6	53
Miscellaneous†	10	43	16	41
	100			

* Could not be classified accurately as to direction of impacts.

† Included 8 collisions with trains; trapped in car, 5; skidding, 4; driver dislocated, 3; side swipe, 2; and double impacts, 3.

Note: There were few mechanical failures: locked steering wheel, 1; steering wheel broke, 1; tire blew out, 2; door accidentally opened while car was under way, 1.

dence of injury to the different parts of the body definitely aid medical attendants in regard to the initial and rapid clinical evaluation of the motorist casualty, whose frequent and multiple injuries pose diagnostic issues that it would be difficult to resolve without this information relative to frequency rates and morbidity.

From the crash-impact point of view, this pattern helps to establish the universality of the forces engendered by automotive design, as shown by the results or effects of accidents, regardless of the type of principal impacts involved in the accidents. This conclusion is in accord with the crash-impact basic idea that impugns automotive design or sources of energy under crash conditions.

The universality of crash force precludes the need of experimental reduplication of *all* the various types of vehicular impacts.

The fairly constant pattern of motorist injuries, in regard to body regional distribution and degree of morbidity, is evidence that these effects of crash force are "not a *direct function of crash force*" but probably are related to the various impact areas (injury potentials) that make up interior automotive design (DeHaven).

However, it should not be forgotten that the various impacts, in conjunction with the speeds at impact, determine the magnitudes of the forces involved in the external automotive environment, which in turn determine the survivability in terms of structural deformation that destroys the integrity of the occupant compartment.

BIBLIOGRAPHY

Campbell, Horace E.: Role of safety belt in nineteen auto crashes, Bull. Am. Coll. Surgeons 40:155, 1955.

DeHaven, Hugh: Current Safety Considerations in the Design of Passenger Seats for Transport Aircraft, Crash Injury Research Release, June, 1952.

———: The Site, Frequency and Danger of Injury Sustained by 800 Survivors of Lightplane Accidents, Crash Injury Research, July, 1952.

DeHaven, Hugh, and Hasbrook, A. H.: Shoulder Harness, Its Use and Effectiveness, Crash Injury Research Release, Nov. 1, 1952.

Kulowski, Jacob: Crash injury research, *Canad. Services M. J. 11:761-766, 1955.*

Steindler, Arthur: The Mechanics of Normal and Pathological Locomotion in Man, Springfield, Ill., Thomas, 1935.

Straith, Claire: Automobile injuries, J.A.M.A. 109:940, 1937.

———: Guest passenger injuries, J.A.M.A. 137:348, 1948.

Stuck, W. G.: Fracture of the sternum and *thyroid cartilage,* Am. J. Surg. *38:560, 1937.*

Woodward, Fletcher D.: Medical criticism of modern automotive engineering, J.A.M.A. 138:627, 1948.

Zollinger, Richard W.: Traffic injuries; a surgical problem, A.M.A. Arch. Surg. 70: 694, 1955.

6

Head Injuries: Januslike Problem

Head injuries always have had a special allure for doctors and have constituted a challenge to them. The caveman also was interested in the mechanical aspects of skull fracture; he seemed to know exactly where to put the axe for the best results. Has the modern automobile replaced the axe? In the present series of 661 motorist survivors, 532, or 80 per cent, received some kind of so-called head injury.

Of the 532 persons with "head" injuries, 273 actually involved the head; 259, the face. The respective frequency rates were 41 per cent and 39 per cent. These injuries are a little more frequent in drivers than in front seat passengers. The distribution of these people as a whole according to seating was 220 drivers (41%—20% head, 21% face); 181 front seat passengers (34%—18% head, 16% face); 35 in the back seat (7%—4% head, 3% face); and 96 unclassified passengers (18%—10% head, 8% face) (Fig. 13).

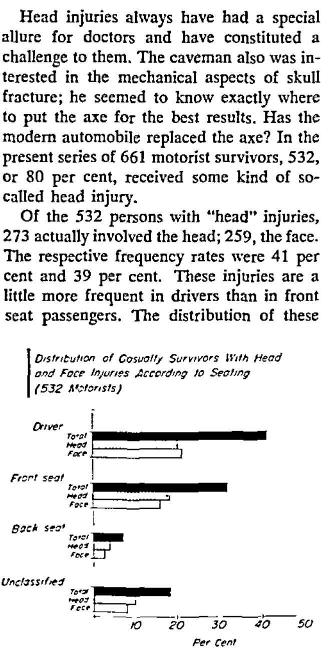


FIG. 13. Ratio and distribution of head and facial injuries in a series of 532 motorist casualty survivors in accordance with seating.

The sexual distribution in head and facial groups is of some interest: for the head there were 3 times as many male as female drivers, and there was a reversal of this ratio favoring women and girls with regard to the right front seat. On the other hand, in those with facial injuries, there were 5 times as many male as female drivers, while the ratio of women to men in the right front seat dropped to 2:1. The sexual distribution in regard to the back seat was about even, and this applied also to the unclassified ones.

FOREHEAD, SCALP, EARS, SKULL AND BRAIN

In this discussion, injuries of the head include the forehead, the scalp, the ears, the skull (vault and base) and the brain. The

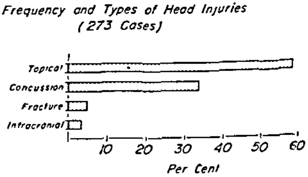


FIG. 14. Nature and ratio of injuries in 273 motorist survivors with head injuries.

TABLE 10. RELATION OF TYPE OF HEAD INJURY TO SEATING IN 273 MOTORIST CASUALTY SURVIVORS

SEATING	NO. OF PERSONS	TOPICAL INJURIES %	SKULL FRACTURE %	CEREBRAL CONCUSSION %	INTERNAL INJURY %	TOTAL NO. INJURIES
Drivers	104	53	5	40	2	173
Right front seat..	94	63	7	26	4	142
Back seat	21	62	8	27	3	26
Unclassified ...	54	66	1	30	3	70
	273					411

3 criteria, established in the pilot study, will be followed throughout the remainder of the clinical section; i.e., topical, fracture and internal injuries.

Of the 273 people with head injuries, there were 244 (59%) with topical lesions; 21 with fractures of the skull (5%); 134 with cerebral concussion (33%); and 12 with various intracranial injuries (3%) (Fig. 14). Of the skull fractures, one third involved the vault, 3 were basal, and the remainder could not be allocated accurately. The relatively low incidence of skull fractures is to be noted, and may be partially accounted for by the fact that head injuries complicated by skull fracture usually are attended by a high mortality rate.

It has been reported (Ulin *et al.*) that in a series of 1,000 head injuries (one third in traffic accidents) 22 per cent of them comprised skull fracture.

In the present series of head injuries, approximately one third manifested various degrees of cerebral concussion. Ulin *et al.* have reported cerebral concussion in 77.5 per cent of their cases with cerebral involvement (570 cases of series of 1,000). The criterion for these was a definite but variable period of post-traumatic unconsciousness or of being "knocked out." From the standpoint of relationship between cerebral concussion and external evidences of head injury, 3 categories were established:

1. Forty-three per cent of these cases did not manifest any kind of external injury.
2. Forty per cent presented lacerations of the scalp, the forehead or the ears.
3. Seventeen per cent exhibited contusions or abrasions of the forehead, the scalp or the ears, alone or in combinations.

Shock was reported to have been present in 21 persons (8%) of this series of head injuries. Eight of them occurred in drivers —12 in the right front seat, 1 in the back seat.

In regard to the seriousness of head injuries, a far more significant picture than the low relative incidence of shock is illustrated by the fact that there were only 32 patients in this series of head injuries (12%) who did not have other concurrent injuries. The remaining 88 per cent manifested multiple injuries.

The type of injury was related to seating in this series of head injuries (Table 10). To begin with, the distribution according to seating in this series was: drivers, 38 per cent; right front seat passengers, 34 per cent; back seat passengers, 8 per cent; unclassified, 21 per cent. About 75 per cent of the drivers were males, and the sexual ratios were reversed in regard to the rest of the occupants. The topical injuries occurred with almost equal frequency in every part of the crash cars. There were more fractures in the right front and back seats than in the driver's seat. Cerebral concussion was more common among driver casualties, but internal injuries were not. However, about 40 per cent of all the injuries were received by drivers.

TABLE 11. NATURE OF HEAD INJURIES RELATED TO THE PRINCIPAL IMPACTS

PRINCIPAL IMPACTS	TOPICAL INJURY %	SKELETAL INJURY %	SHOCK %	CEREBRAL CONCUSSION %	INTERNAL INJURY %
Forward vehicular collisions	54	8	5	30*	3
Rear-end collisions	56	11	0	22	11
Angular collisions (from side)	58	0	0	26	16
Collision and roll-over	63	0	0	37	0
Collision with ejection of occupant..	48	3	0	42	6
Forced off the road	40	0	10	50	0

* Includes 2 postconcussive psychoses

TABLE 12. TYPE AND FREQUENCY OF FACIAL INJURIES IN 295 MOTORIST CASUALTY SURVIVORS

NATURE OF INJURY	No.	%
Laceration	203	54
Contusion and laceration	91	24
Fracture	80	22
	374	100

Table 11 relates the type of head injuries to the principal impacts.

Rear-end collisions seemed to play a larger role in head injuries than in facial injuries. Again, topical injuries were about equally distributed in regard to all kinds of accidental impacts. Shock occurred only from forward and off-the-road accidents. Interestingly enough, cerebral concussion was a common denominator for all kinds of accidental impacts. The highest incidence of internal injuries occurred from rear end and angular collisions from the side.

THE FACE

Inclusive of lesions of the forehead (included for statistical distribution) there were 295 persons (45% of the entire series of 661 motorist-casualty survivors) with facial injuries, a frequency second only to that of the extremities The type and the over-all frequency of these injuries were found to be: lacerative, 54 per cent; contusive and abrasive, 24 per cent; and fracture, 22 per cent (Table 12). The large number of lacerative lesions of the face is to be remarked. The frequency of injuries could be classified according to the different areas of the face, i.e., upper one third, middle one third and lower one third. Of the 333 lesions that could be allocated thus, it was found that 33 per cent involved the upper third of the face, 53 per cent, the middle third; and 14 per cent, the lower one third (Fig. 15).

Fifty-three injuries involved the eye areas, several of which were quite severe. Of these, the conjunctival tissues often were involved; the cornea, rarely, and this was associated most frequently with fractures of the zygoma, especially when this was complicated by injury to the infra-orbital nerve. Twenty-seven people had lacerations involving the lips; 17 had lacerations of the external and the in-

TABLE 13. FREQUENCY OF FRACTURES OF THE FACIAL BONES*

AREA OF FACE AFFECTED	BONES AFFECTED	No.	%
Upper third.........	Frontal	7	6
Middle third	Nasal	26	21
	Zygoma	22	19
	Maxilla	14	12
Lower third.	Mandible	30	25
		99	83

* The teeth were involved in 19 persons (17%)

ternal nares. Exclusive of injuries to the teeth, 12 had intra-oral lacerations, in one of which the tongue was cut, and in another, the uvula. The vulnerability of the middle one third of the face to crash forces in motorists has been reaffirmed, fractures in the various bones of the face (including the forehead) being frequent (Table 13).

Several of those with fractures of the facial bones received bilateral injuries, especially those with mandibular ones. Fractures were compounded in 3 nasal, 1 maxillary and 2 mandibular injuries. Undoubtedly, there were a few more of the last. The teeth were driven into the maxillary sinus in only 1 instance; however, the teeth were involved in at least 19 persons, and more were lost afterward. Concurrent fractures in this group of facial ones included: 19 of the extremities, 13, the chest; 3, the skull; 2, the spine; and 2, the pelvis. Eleven of these patients exhibited varying degrees of shock; 18 manifested cer-

TABLE 14. RELATION OF NATURE OF FACIAL INJURIES TO THE PRINCIPAL VEHICULAR IMPACTS

PRINCIPAL IMPACTS	NO. OF SOFT TISSUE INJURIES	NO. OF FRACTURES	TOTAL NO. OF CRASHES
Collision with other vehicles.	24	12	36
Head-on collisions	17	6	23
Forcibly ejected from vehicle..	13	1	14
Collision with fixed object...	9	2	11
Roll-over	7	3	10
Angular collisions (from side) ..	6	3	9
Ran off road....	7	1	8
Lost control (blowout) ...	0	2	2
Miscellaneous ..	4	0	4
	87	30	117

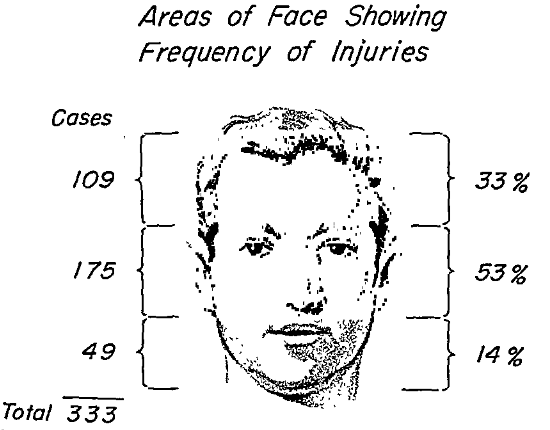


FIG. 15. Distribution of facial injuries in a series of 295 motorist casualty survivors according to the various facial levels, including forehead.

Facial Fractures According to Seating

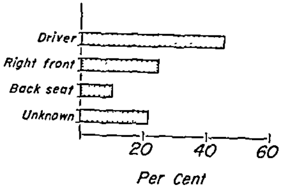


FIG. 16. Graphic representation of distribution of facial injuries (295 survivors) according to seating.

ebral concussion; and 2 had concurrent internal injuries—1 head and 1 chest.

The effect of relationship of occupants to impact areas as a predisposing cause of injury has been well established in regard to fracture-producing impacts. Of 121 drivers, 37 (31%) received fractures. In addition to this, there were 96 right front seat passengers, 18 of whom (19%) had fractures. From the standpoint of number of persons with fractures, 69 per cent occurred in the front seat. Of 27 persons in the back seat, 9 (33%) had fractures; and, of the 51 unclassified persons, 16 (31%) received fractures. The average percentage from this standpoint was 27 per cent (Fig. 16).

The soft tissue and fracture injuries are related to the principal vehicular impacts, the vast majority of which were forward ones (Table 14). It was found that all types of accidents resulted in approximately similar rates of soft tissue and fracture injuries. However, in the forcible ejection group, all facial injuries were soft tissue types, with only 1 fracture occurring in 14 people ejected forcibly. The average rate for soft tissue and fracture injuries for all types of impacts was 74 and 26 per cent, respectively.

The 10-pound head inflicts severe damages upon the face in forward types of impacts. The relative looseness of the overlying skin predisposes this area to lacerations. The malar bones and others buffer energy absorption that otherwise might go to the skull. The mobility of the mandible protects the front of the neck and moderates energies in its own behalf.

COMMENT: HEAD AND FACE

Orthopaedic surgeons hardly need to be reminded that all complicated head injuries should be in the hands of a neurosurgeon. Gurdjian, Lissner *et al.* have established some interesting biomechanical factors in regard to skull fractures: (1) overlying soft parts absorb energy that is bufferlike in its action; (2) linear fractures are the result of excessive tensile stresses; (3) for practical purposes of engineering design of automotive interiors, the limits of elasticity of the skull is about 400 inch pounds; (4) once this limit has been breached, explosive effects are likely to follow; and (5) the fracture site may be predicted when the impact area is known.

The relationships between the elastic skull and the incompressible contents (that is, under conditions of abrupt accelerations or decelerations) make this an excellent situation for increased intracranial pressures during rapid changes of velocity of the head. Concussion is supposed to result when the intracranial pressure gets above the systolic blood pressure.

The relationships between injury and cerebral concussion have received considerable attention because the living human skull-force situation is so unique in that tensile and compressive deformations must take place in response to applied external forces through deformation-dissipation processes, limited and/or governed by the fact that any deformation of the skull will be resisted by a concurrent rise in intracranial pressure. The rate of change in velocity, not the momentum (Denny-Brown), is the critical factor in the production of cerebral concussion; from zero to 28 ft. per second is sufficient. Both

direct and indirect violence may produce concussion, although cranial restitution is facilitated when the head is in motion, not fixed. During motorist accidents a combination of compression, deceleration and acceleration forces frequently co-exists and produces concussion. Most recently, Gurdjian *et al.* have attributed cerebral concussion to a derangement in function of the brain stem, i.e., from injury to that part of the brain from increased intracranial pressure at the time of impact, direct injury by distortion, mass movement, shearing or destruction by a missile.

On the clinical side these authors stated some years ago that (1) there was a combination of pathologic processes inside the head in all fatal cases; (2) there was always gross evidence of this; (3) massive hemorrhage usually was on top of the brain and, therefore, amenable to surgical treatment; (4) epidural hemorrhage should be searched for in the fracture line; and (5) subdural hemorrhage often was opposite the gross fracture line.

MANAGEMENT: FACIAL INJURIES

The initial basic needs are for compressive dressings, open airway and supportive dressing for the jaw, if indicated.

The emergency treatment is made more effective after a routine but thorough examination with regard to the following anatomic areas: note all lacerations; check for abnormal contours of the facial bones (especially the malar and the zygomatic arches); look into the nasal cavity for obstructions and compounding of fractures; examine the inside of the mouth for loose teeth, lacerations and also evidences of fracture compounding.

Needless to say, adequate roentgenographic examinations should be made as soon as possible, since most facial bones are quite easy to reduce if done soon after injury. Special technics are required to detect asymmetries of the optic foramina and other lesser displacements. The latter should be looked for in all depressed fractures of the malar and the maxillary bones. Fractures of the mandible often are placed asymmetrically; i.e., subcondylar on one side and at the angle on the other. Also, in all malar fractures, a careful check for corneal injuries and infraorbital nerve damage always should be made. Moreover, it should be noted carefully whether malar or zygomatic injuries interfere in any way with mandibular movements. Finally, the malar or other facial fracture that is complicated by double or other disturbances of vision should be subjected to the most careful scrutiny for cause by competent medical authority. In 1 case of the author's there was a depressed fracture of the dorsum sellae which complicated a depressed malar fracture. A decompressive operation restored this patient's vision.

The definitive care of facial fractures calls for some versatility on the part of those undertaking it. The vast majority of facial fractures will respond to more or less conservative methods of treatment. It is necessary to elevate depressed malar and zygomatic arch fractures. The former can be approached directly or indirectly; the latter always can be hooked and pulled outward into place. Maxillary fractures usually can be controlled by traction devices fixed to the teeth and a headpiece. Mandibular fractures do well when interdental wiring can be employed. Most of these will get well with elastic plaster chin and headpieces, or similar devices. Dental consultation always is in order. Open reduction is rarely needed.

Residual treatment refers as a rule to plastic repair. This emphasizes the care called for in the closure of all facial lacerations, however trivial these may seem to be against the background of other concurrent injuries at the time the patient is admitted to the hospital. Facial scars are the most persistent residuals of motorist injuries in survivors. For these reasons, the primary care of facial injuries should follow certain general principles aimed at minimizing scars.

CONCLUSIONS

The frequency of head and facial injuries have called attention to the injury potentials of windshield, dash and steering controls. Prophylactic measures include the following possibilities: revision of interior surfaces to promote controlled and progressive deformation; revised safety glass with controlled deformation characteristics; safety restraints for occupants; changes in spatial relationships between occupants and impact areas; the wearing of a face mask and/or protective helmet. Any measure that will protect the face will also have a sparing action on the head in forward impacts. The converse is not true.

Since head injuries that are complicated should be cared for by a neurosurgeon, the transport of the patient to that specialist should be undertaken at once. During transport, the usual humanitarian splinting of broken extremities is undertaken. The patient should be rolled over on his side and the airway kept open. Signs of cerebral concussion should be watched for. Head injuries tolerate transport very well under these conditions. There seems to be no relation between the severity of the cerebral damage and linear skull fracture; people may die from concussion alone.

The prevention of scarring is one of the gravest responsibilities of those who undertake to treat facial injuries.

Together, head and facial injuries are the most frequent ones encountered in motorist-casualty survivors Their separate considerations give an entirely different picture of "head" injuries from that previously supposed to be the case.

BIBLIOGRAPHY

Blocker, T. G., Jr.: Primary care of injuries about the face, J.A.M.A. 159:422, 1955.

Cornell Aeronautical Laboratory, Inc.: Head Impact Research, Final Report, Jan., 1948.

Denny-Brown, D.: Concussion of the brain, Brain 64:98, 1941.

Evans, J. P.: *Acute Head Injury*, Springfield, Ill., Thomas, 1950.

Gerber, S. R.: An analysis of fatal accidents in Guyahoga County for a period of three years, Ohio State M. J. 37:17, 1941.

Gurdjian, E. S., and Webster, J. E.: Acute head injuries, J.A.M.A. 134:1072, 1947.

Gurdjian, E. S., Webster, J. E., and Lissner, H. R.: Observations on the mechanism of brain concussion, contusion and laceration, Surg., Gynec. & Obst. 101:680, 1955.

———: Studies on skull fractures with particular reference to engineering factors, Am. J. Surg. 78:736, 1949.

Ivy, R. H., and Curtiss, L.: Fractures of the Jaws, ed. 2, Philadelphia, Lea & Febiger, 1938.

Kazanjian, V. H., and Converse, J. M.: The Surgical Treatment of Facial Injuries, Baltimore, Williams & Wilkins, 1949.

Kulowski, Jacob: Accidental head injuries in occupants of crash motor vehicles, Am. Surgeon, in press.

———: Motorist injuries of the face, J.A.D.A., in press.

Rowbottom, G. F.: *Acute Injuries to the Head*, Edinburgh, Livingstone, 1942.

Scott, W. W.: Physical concussion, Arch. Neurol. & Psychiat. 43:270, 1940.

Souttar, H. S.: Physics and the Surgeon, Oxford, Blackwell, 1948.

Ulin, A. W., Rosomoff, H. L., Berkowitz, D., and Olsen, A. K.: Acute head injuries: a review of 1,000 cases, Am. Surgeon 19:226, 1953.

7

Injuries of Chest and Abdomen

Among the total series of 661 motorist-casualty survivors, 216 (33%) received injuries of the chest and the abdomen (28% and 5% respectively). Of the latter, 181 (84%) involved the chest and 35 (16%) the abdomen. There were 117 males and 99 females. All decades through the ninth were represented (Fig. 17). Fifty-eight per cent of these people were drivers (Table 15). About 75 per cent of these were males, but 75 per cent of the right front seat passengers were females. The large number of contusive lesions as against the paucity of abrasive and lacerative ones (Table 16) is to be noted, as is the relatively low incidence of internal injuries. Approximately 50 per cent of the total number of injuries of the chest were fractures (Table 17).

Ninety-five persons with chest injuries suffered fractures of the ribs (Table 17). Of these, 21 per cent were solitary ones; 28 per cent, double fractures; and 50 per cent received 3 or more rib fractures. The left side of the chest predominated in all kinds of fractures except the double ones, probably because right-handed people turn instinctively to the right to avoid injury. Of the multiple fractures, 8 were bilateral The number of fractures of the first rib (14) were notable, and almost always they were associated with fracture of the clavicle. Not so long ago, injuries to this rib were considered to be rarities. At the other extreme, the low incidence of fractures of the last 2 ribs is to be remarked. The vulnerability of the middle one third of the thoracic cage is comparable with the injury incidence of the middle one third of the face (Fig. 18). In addition to the rib fractures, there were 9 fractures of the sternum—4 solitary and 5 associated with other thoracic lesions. Six of these occurred in drivers, 2 in right front seat guest passengers, and 1 could not thus be classified.

Other complicating injuries are to be noted in Table 18.

TABLE 15. DISTRIBUTION OF OCCUPANTS WITH INJURIES OF THE CHEST AND THE ABDOMEN ACCORDING TO SEATING

SEATING	NO.	%
Driver	125	58
Right front seat	54	25
Back seat	11	5
Unclassified	26	12

CLINICAL—ABDOMEN

Internal injuries of the abdomen included: ruptured spleen, 2; ruptured gallbladder, 1; eventration of the diaphragm, 1; kidney rup-

TABLE 16. FREQUENCY AND NATURE OF CHEST AND ABDOMINAL INJURIES IN 216 MOTORIST CASUALTIES

TYPE OF LESION	NO. OF LESIONS CHEST	NO. OF LESIONS ABDOMEN	TOTAL NO.	TOTAL %
Contusion	94	29	123	46
Abrasion	4	5	9	3
Laceration	4	1	5	2
Skeletal	113	0	113	42
Internal	10	10	20	7
	225	45	270	100

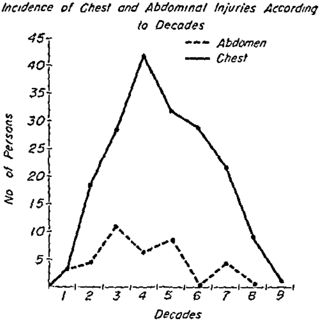


FIG 17. Graphic representation of incidence of chest and abdominal injuries according to decades in a series of 216 motorist casualties (survivors discussed in text). The tolerance of the abdominal areas to crash forces is apparent from the relatively low incidence of abdominal injuries as compared with the higher incidence of chest injuries. In other words, the chest is more vulnerable than the abdomen under crash conditions and/or upsets

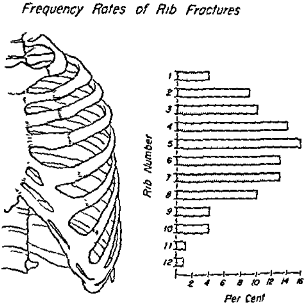


FIG. 18. Graphic representation of rate of fracture of the various ribs in a series of 181 motorist casualty survivors. In silhouette the graph indicates that the most prominent part of the chest wall (like the middle third of the face) is most vulnerable to crash forces (assuming the graph represents the lateral view of the rib cage). The relative frequency of fractures of the upper ribs is to be noted, the converse is true in regard to the lowermost ones.

TABLE 17. FREQUENCY RATES OF VARIOUS RIB FRACTURES AMONG 181 SURVIVORS WITH CHEST INJURIES

Ribs Affected	Solitary Rib		Double Ribs		Multiple Ribs		Total No.		Total	
	Right	Left	Right	Left	Right	Left	Right	Left	No.	%
1st	1	1	1	1	7	3	9	5	14	4.0
2nd	2	1	1	4	8	13	11	18	29	9.0
3rd	2	0	3	3	12	13	17	16	33	10.0
4th	0	1	5	5	15	19	20	25	45	14.0
5th	0	1	6	4	19	23	25	28	53	16.0
6th	0	3	3	3	12	22	15	28	43	13.0
7th	1	0	4	2	7	18	12	20	42	13.0
8th	1	0	1	0	7	13	9	13	32	10.0
9th	0	1	1	2	5	6	6	9	15	4.0
10th	1	2	4	2	3	2	8	6	14	4.0
11th	0	1	2	1	0	0	2	2	4	2.0
12th	0	2	0	0	0	0	0	2	2	1.0
	8	13	31	27	95	132	134	172	326	100 0

In addition to the above tabulated cases, 8 patients were said to have had "multiple rib fractures." Of the double rib fracture cases above, 6 were bilateral. In 1 case, there was an isolated rib fracture on each side. In 3 of this group, there were double rib fractures on 1 side and solitary on the other



FIG. 19. (*Top*) Appearance of young male driver about 2 weeks after impaction of steering controls—progressively increasing abdominal pain and loss of weight. (*Bottom*) Same patient, showing palpable mass in epigastrium, which proved to be blood and clot in the lesser omentum, probably from laceration into ligament of Treitz.

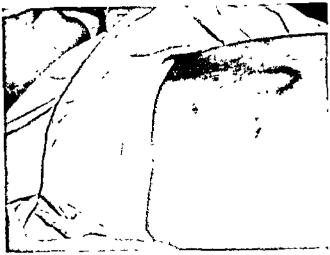


FIG. 20. Showing contusion right midanterior chest wall due to driver impact onto the steering post of a 1954 passenger car. Collision was high speed, with truck, forward in type. Patient had had about 4 other motorist accidents in the past (1 eye lost after one and subsequent grand mal attacks after 2 of the others). Patient saw inevitability of the crash, covered his face with his arms, turned slightly to the left and impacted the steering controls in this fashion. Received large laceration of the forehead (visor?), contusion right anterior chest, fracture 3rd through 6th ribs (right anterior axillary line), contusion sprain spine and shock.

ture, 2; kidney contusion, 3; and mesenteric tear (probably ligament of Treitz) with hemorrhage into lesser omental sac, 1. In 4 cases, internal lesions were suspected as follows: bleeding of unknown origin from the upper gastro-intestinal tract, large bowel obstruction and ileus.

Some of the pitfalls in the diagnosis of acute intra-abdominal lesions were especially notable in 2 cases of this series. In both patients, the diagnosis was not confirmed until 2 weeks after injury. Both were young male drivers who had impacted their steering controls in high speed forward collisions. Their severe multiple injuries on admission masked their abdominal complaints for some time.

The first patient went downhill due to abdominal pain and marked loss of weight. On readmission (Fig. 19) a pancreatic injury was suspected. Emergency operation disclosed compressive clotted blood in the lesser omentum. Recovery followed evacuation and drainage. The second man was found to have an eventration of the diaphragm on readmission. A case with ruptured gallbladder, due to impaction of the steering wheel, was operated upon without complications. Two ruptured spleens were removed: in 1, the postoperative course was smooth; in the other—the patient was not seen until the third day after injury—there was the complication of bronchogenic exudates that required tracheotomy and aspirations for survival.

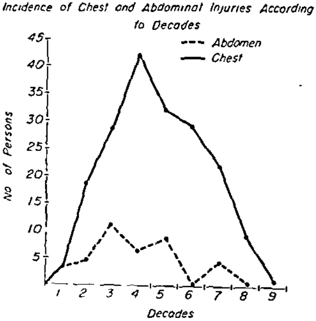


FIG. 17. Graphic representation of incidence of chest and abdominal injuries according to decades in a series of 216 motorist casualties (survivors discussed in text). The tolerance of the abdominal areas to crash forces is apparent from the relatively low incidence of abdominal injuries as compared with the higher incidence of chest injuries. In other words, the chest is more vulnerable than the abdomen under crash conditions and/or upsets.

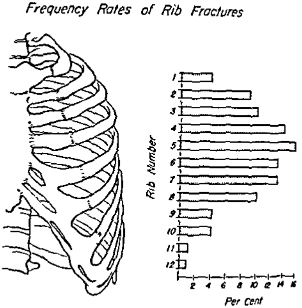


FIG. 18. Graphic representation of rate of fracture of the various ribs in a series of 181 motorist casualty survivors. In silhouette the graph indicates that the most prominent part of the chest wall (like the middle third of the face) is most vulnerable to crash forces (assuming the graph represents the lateral view of the rib cage). The relative frequency of fractures of the upper ribs is to be noted; the converse is true in regard to the lowermost ones.

TABLE 17. FREQUENCY RATES OF VARIOUS RIB FRACTURES AMONG 181 SURVIVORS WITH CHEST INJURIES

Ribs Affected	Solitary Rib		Double Ribs		Multiple Ribs		Total No.		Total	
	Right	Left	Right	Left	Right	Left	Right	Left	No.	%
1st	1	1	1	1	7	3	9	5	14	4.0
2nd	2	1	1	4	8	13	11	18	29	9.0
3rd	2	0	3	3	12	13	17	16	33	10.0
4th	0	1	5	5	15	19	20	25	45	14.0
5th	0	1	6	4	19	23	25	28	53	16.0
6th	0	3	3	3	12	22	15	28	43	13.0
7th	1	0	4	2	7	18	12	20	42	13.0
8th	1	0	1	0	7	13	9	13	32	10.0
9th	0	1	1	2	5	6	6	9	15	4.0
10th	1	2	4	2	3	2	8	6	14	4.0
11th	0	1	2	1	0	0	2	2	4	2.0
12th	0	2	0	0	0	0	0	2	2	1.0
	8	13	31	27	95	132	134	172	326	100.0

In addition to the above tabulated cases, 8 patients were said to have had "multiple rib fractures." Of the double rib fracture cases above, 6 were bilateral. In 1 case, there was an isolated rib fracture on each side. In 3 of this group, there were double rib fractures on 1 side and solitary on the other.



FIG. 19. (*Top*) Appearance of young male driver about 2 weeks after impaction of steering controls—progressively increasing abdominal pain and loss of weight. (*Bottom*) Same patient, showing palpable mass in epigastrium, which proved to be blood and clot in the lesser omentum, probably from laceration into ligament of Treitz.

ture, 2; kidney contusion, 3; and mesenteric tear (probably ligament of Treitz) with hemorrhage into lesser omental sac, 1. In 4 cases, internal lesions were suspected as follows: bleeding of unknown origin from the upper gastro-intestinal tract, large bowel obstruction and ileus.

Some of the pitfalls in the diagnosis of acute intra-abdominal lesions were especially notable in 2 cases of this series. In both patients, the diagnosis was not confirmed until 2 weeks after injury. Both were young male drivers who had impacted their steering controls in high speed forward collisions. Their severe multiple injuries on admission masked



FIG. 20. Showing contusion right mid-anterior chest wall due to driver impact onto the steering post of a 1954 passenger car. Collision was high speed, with truck, forward in type. Patient had had about 4 other motorist accidents in the past (1 eye lost after one and subsequent grand mal attacks after 2 of the others). Patient saw inevitability of the crash, covered his face with his arms, turned slightly to the left and impacted the steering controls in this fashion. Received large laceration of the forehead (visor?), contusion right anterior chest, fracture 3rd through 6th ribs (right anterior axillary line), contusion sprain spine and shock.

their abdominal complaints for some time.

The first patient went downhill due to abdominal pain and marked loss of weight. On readmission (Fig. 19) a pancreatic injury was suspected. Emergency operation disclosed compressive clotted blood in the lesser omentum. Recovery followed evacuation and drainage. The second man was found to have an eventration of the diaphragm on readmission. A case with ruptured gallbladder, due to impaction of the steering wheel, was operated upon without complications. Two ruptured spleens were removed: in 1, the postoperative course was smooth; in the other—the patient was not seen until the third day after injury—there was the complication of bronchogenic exudates that required tracheotomy and aspirations for survival.

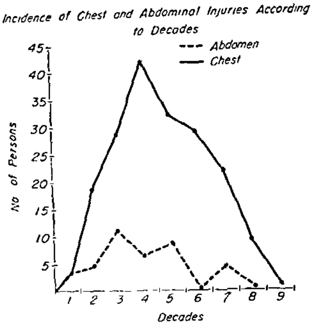


FIG. 17. Graphic representation of incidence of chest and abdominal injuries according to decades in a series of 216 motorist casualties (survivors discussed in text). The tolerance of the abdominal areas to crash forces is apparent from the relatively low incidence of abdominal injuries as compared with the higher incidence of chest injuries. In other words, the chest is more vulnerable than the abdomen under crash conditions and/or upsets.

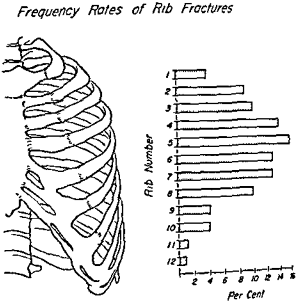


FIG. 18. Graphic representation of rate of fracture of the various ribs in a series of 181 motorist casualty survivors. In silhouette the graph indicates that the most prominent part of the chest wall (like the middle third of the face) is most vulnerable to crash forces (assuming the ... of the rib ... verse is true in regard to the lowermost ones.

TABLE 17. FREQUENCY RATES OF VARIOUS RIB FRACTURES AMONG 181 SURVIVORS WITH CHEST INJURIES

RIBS AFFECTED	SOLITARY RIB		DOUBLE RIBS		MULTIPLE RIBS		TOTAL NO.		TOTAL	
	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	NO.	%
1st	1	1	1	1	7	3	9	5	14	4.0
2nd	2	1	1	4	8	13	11	18	29	9.0
3rd	2	0	3	3	12	13	17	16	33	10.0
4th	0	1	5	5	15	19	20	25	45	14.0
5th	0	1	6	4	19	23	25	28	53	16.0
6th	0	3	3	3	12	22	15	28	43	13.0
7th	1	0	4	2	7	18	12	20	42	13.0
8th	1	0	1	0	7	13	9	13	32	10.0
9th	0	1	1	2	5	6	6	9	15	4.0
10th	1	2	4	2	3	2	8	6	14	4.0
11th	0	1	2	1	0	0	2	2	4	2.0
12th	0	2	0	0	0	0	0	2	2	1.0
	8	13	31	27	95	132	134	172	326	100.0

In addition to the above tabulated cases, 8 patients were said to have had "multiple rib fractures." Of the double rib fracture cases above, 6 were bilateral. In 1 case, there was an isolated rib fracture on each side. In 3 of this group, there were double rib fractures on 1 side and solitary on the other.





FIG. 19. (*Top*) Appearance of young male driver about 2 weeks after impaction of steering controls—progressively increasing abdominal pain and loss of weight. (*Bottom*) Same patient, showing palpable mass in epigastrium, which proved to be blood and clot in the lesser omentum, probably from laceration into ligament of Treitz.



FIG. 20. Showing contusion right midanterior chest wall due to driver impact onto the steering post of a 1954 passenger car. Collision was high speed, with truck, forward in type. Patient had had about 4 other motorist accidents in the past (1 eye lost after one and subsequent grand mal attacks after 2 of the others). Patient saw inevitability of the crash, covered his face with his arms, turned slightly to the left and impacted the steering controls in this fashion. Received large laceration of the forehead (visor?), contusion right anterior chest, fracture 3rd through 6th ribs (right anterior axillary line), contusion sprain spine and shock.

ture, 2; kidney contusion, 3; and mesenteric tear (probably ligament of Treitz) with hemorrhage into lesser omental sac, 1 In 4 cases, internal lesions were suspected as follows: bleeding of unknown origin from the upper gastro-intestinal tract, large bowel obstruction and ileus.

Some of the pitfalls in the diagnosis of acute intra-abdominal lesions were especially notable in 2 cases of this series. In both patients, the diagnosis was not confirmed until 2 weeks after injury. Both were young male drivers who had impacted their steering controls in high speed forward collisions. Their severe multiple injuries on admission masked their abdominal complaints for some time.

The first patient went downhill due to abdominal pain and marked loss of weight. On readmission (Fig. 19) a pancreatic injury was suspected. Emergency operation disclosed compressive clotted blood in the lesser omentum. Recovery followed evacuation and drainage. The second man was found to have an eventration of the diaphragm on readmission. A case with ruptured gallbladder, due to impaction of the steering wheel, was operated upon without complications. Two ruptured spleens were removed: in 1, the postoperative course was smooth; in the other—the patient was not seen until the third day after injury—there was the complication of bronchogenic exudates that required tracheotomy and aspirations for survival.

TABLE 18. COMPLICATING LESIONS AMONG 95 SURVIVORS WITH RIB FRACTURES

NATURE OR AREA OF COMPLICATION	No.	%
Head	39	21.0
Extremities	50	27.0
Multiple topical lesions	33	18.0
Cerebral concussion	17	9.0
Shock	11	7.0
Intrathoracic	10	7.0
Abdominal	8	4.0
Trunk	7	3.0
Neck	4	2.0
Pelvis	4	2.0
	183	100.0

In a general way, the greater the number of fractured ribs, the greater was the number of concurrent complicating injuries. The chief exception to this was in regard to the head, wherein solitary rib fractures were associated with more head injuries than double ones.

TABLE 19. PRINCIPAL IMPACTS RELATED TO CHEST INJURIES

IMPACTS	NO. OF INJURIES	%
Forward vehicular collisions	32	38
Roll-over	11	13
Collision with fixed object	10	12
Impact from side	10	12
Ejected forcibly	7	8
Ran off road	4	5
Hit by train	3	4
Rear-end collision	3	4
Lost control	3	4
	83	100

All motorists injuries (those injured by external forces generated within the occupant compartment) attest to what the author has termed the universality of force; that is, the force is the same under all conditions of crash but differs in its effects by virtue of various physical factors that govern injury (direction, intensity, area of pressure, duration, velocity at onset). This was reflected in the fact that all kinds of injuries were produced similarly by the various principal impacts.

CLINICAL—CHEST

Tables 19 and 20 illustrate the principle of universality of force in regard to chest injuries in general and rib fractures, respectively. The general patterns are quite similar, regardless of the nature of the impacts (Fig 20); but, statistically, the results do differ because certain types of impacts occur more frequently than others.

Above all others, chest injuries demand a diagnostic and therapeutic versatility that few medical attendants possess completely. The liberal use of internists and thoracic surgeons is recommended strongly to help diagnose and treat the various primary injuries and—what is even more challenging—the serious nature of the complications. Mediastinal injuries comprise an even more unique category from this standpoint, as has been pointed out in the chapter on pathology.

Cardiac arrest may result from tamponade, contusion or laceration of the heart itself. Baseline EKG and adequate roentgenography on admission to the hospital would facilitate the diagnosis of such injuries. Such conditions which interfere with cardiac muscular action (tamponade or laceration of the pericardium) can be recognized most readily on fluoroscopic rather than roentgenographic films alone. Mediastinal injury always should be suspected in face of any "stove-in" chest; failure of a "pneumothorax" to respond to aspiration; and any unexplained abnormal shifts or other displacements in the chest cavity.

Requisites for survival demand a stable chest wall (without paradoxic breathing) (Fig. 21); an airtight chest wall (without tension pneumothorax); open airways (free from exudates; tracheotomy reduces dead space of upper airway by about 150 cc. and facilitates aspiration of secretions); and an adequate circulating blood volume.

EXPERIMENTAL OBSERVATIONS

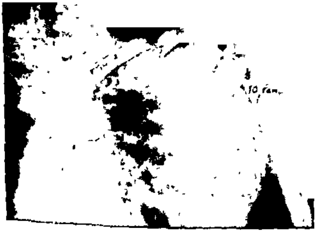
The remarkable tolerances of the chest and the abdomen to linear decelerative forces

TABLE 20. NATURE OF INJURY OF THE CHEST RELATED TO PRINCIPAL IMPACTS

PRINCIPAL IMPACTS	NO. OF SOFT TISSUE INJURIES (EXTERNAL)	NO. SINGLE RIB FRACTURES	NO. DOUBLE RIB FRACTURES	NO. MULTIPLE RIB FRACTURES	TOTAL NO.	TOTAL %
Forward vehicular collisions	9	9	5	9	32	38
Roll-over	5	2	0	4	11	13
Collision with fixed object	2	0	3	5	10	12
Impact from side	4	1	2	3	10	12
Ejected forcibly	4	2	0	1	7	8
Ran off road	1	0	1	2	4	5
Hit by train	1	0	0	2	3	4
Rear end collision	2	1	0	0	3	4
Lost control	1	0	2	0	3	4
	29	15	13	26	83	100

This illustrates the universality of injury-producing forces. However, variable effects were reflected in the following: in 29 impacts there were only external (topical) injuries; in 15 cases, solitary rib fractures; in 13 cases, double rib fractures; and in 26 cases, multiple rib fractures.

under controlled experimental conditions are of great significance in aiding survival in aircraft and ground vehicle crashes. This ability of the human body to withstand high external forces has been established largely through various methods of loadings (static



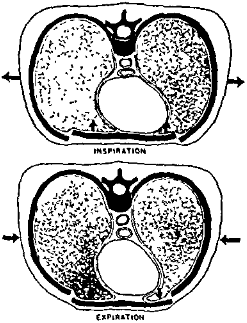


FIG. 21. Roentgenogram showing fractures of the 1st through the 7th ribs and the clavicle (*left*). This man manifested a so-called paradoxic breathing, which is to be expected from any completely divorced section of the chest wall, especially when it happens to be the sternum (see *right*). However, peculiar types of breathing mechanics may result from upper (and other) thoracic skeletal injuries also; occasionally, for unexplained reasons, voluntary or reflex. Always suspect a mediastinal injury! It has been stated also that even 1 fractured rib may reduce the respiratory exchange by 10 per cent. (*Above*) Shows mechanism of paradoxic breathing, i.e., inspiration draws isolated section inward, expiration does the opposite.

or dead and dynamic or live) to the chest and the abdomen.

Goldman has made estimates showing that the chest can tolerate in excess of 600 pounds before respiratory distress is acute and in excess of 1,000 pounds for approximately 10 milliseconds before pain is acute.

The remarkable tolerance of a properly suspended human body to withstand linear decelerative forces has been demonstrated most conclusively by the amazing self experimental observations of Stapp. Severy has charted the decelerative patterns of automobile structure, plane fuselage and Stapp's sled decelerative patterns. Thus presented, the relatively brief human decelerations stand out sharply.

In DeHaven's series of 800 survivors, there were only 23 intra-abdominal injuries, with bruising where the belts had been worn.

Two exceptions to these reports, favorable to safety gear, should be mentioned:

Teare, from postmortem data on plane-crash victims, reported that "the immediate cause of death in more than one half of the victims (Viking crash) was acute flexion of the body over the safety belt." DuBois re-examined Teare's report. He found that these injuries were quite typical, that they already had been reported by CIR, and that they were, therefore, not caused by the seat belts.

The second exception to the rule of great tolerances to impacts of the chest and the abdomen was reported by the American Society of Safety Engineers (ASSE), who were concerned with the effects of safety equipment ordinarily worn by window washers and the like. It was evident that these experiments were inconclusive in so far as the results were concerned in relation to crash-barrier equipment ordinarily worn by pilots.

Mechanical tests made by the ASSE were of interest also. These showed that it required a magnitude of 22 pounds to cause fracture of isolated ribs, which distorted on the average of 22 6 per cent before fracture occurred. Olsen machine tests on halves of thoraces (cadaver) also were made. Intact cadaver thoraces took forces greater than 5,500 pounds through a 4-in. belt before single rib fractures occurred. On the other hand, a deviscerated thorax could not be fractured by the belt, but its distortion was so great under such loading that the sternum could be forced down against the spinal column.

CONCLUSIONS

Injuries of the chest and the abdomen present one of the many seemingly paradoxic situations that abound in the field of motorist-casualty survivors. Chest injuries are frequent and dangerous; abdominal injuries are infrequent and dangerous. Therefore, these regions have a common denominator — dangerousness.

A more significant similarity derives from the great capabilities of both to absorb tremendous decelerative forces when these are transmitted to a properly suspended body by safety gear. The argument for safety restraint of vehicular occupants has been clinched by Colonel Stapp's epochal demonstration of the body's physiologic tolerances to great g forces in this manner. The psychological and the social implications of this have not yet been grasped by the public in regard to the reduction of injuries in crashes.

From the clinical angle, abdominal internal injuries rarely present any real problems of treatment after the diagnosis has been made. On the other hand, crushing injuries of the chest (and even lesser ones) always present problems of both diagnosis and treatment. It is suggested that internists be made a part of the diagnostic and therapeutic team in these cases, especially since these become more medical as the interval of survival increases.

The apparent increase in chest injuries must inevitably increase the frequency of mediastinal injuries. Thus, primary shock reaches its zenith of importance and clinical attraction in situations far removed from wound shock as it has been commonly understood.

It is becoming increasingly evident that prophylaxis relative to motorist injuries must be focused ever more on the prevention of chest injuries if the immediate mortality is to be reduced from crashes.

BIBLIOGRAPHY

Bierman, H. R.: The protection of the human body from impact forces of fatal magnitude, Mil. Surgeon 100:125-141, 1947.

———: Protection of the human body; principles as applied in a restraining harness for aircraft pilots, J.A.M.A. 133:552, 1947.

———: Some physical principles in reporting on aircraft crashes, Bureau of Medicine News Letter (Aviation Supp.) 6:3-5, 1946.

Blades, Brian: Physiologic principles underlying the treatment of injuries to the chest, Am. J. Surg. 78:665, 1949.

Cameron, D. A., *et al.*: An analysis of management and complications of multiple rib fractures (3 or more), Am. J. Surg. 78:668, 1949.

DuBois, E. F.: Safety belts are not dangerous, Brit. M. J. 3:685, 1952.

Goldman, D.: Mechanical forces acting on aviation personnel, J. Aviation Med. 17:426, 1946.

Heckel, Eileen, *et al.*: Cardiac arrest during surgical operations, S. Clin. North America 35:243, 1955.

Heroy, W. W., *et al.*: Method of skeletal traction through the sternum in "steering wheel" injury to the chest, Ann. Surg. 133:135, 1951.

Holinger, P. H., *et al.*: Tracheotomy, S. Clin. North America 35:21, 1955.

Kulowski, J., and Ryan, J. H.: Isolated bilateral fracture of the first rib (Horner's), South. M. J. 33:1149, 1940.

Mayer, John H.: Fatal Chest Injuries, Paper Given Before the National Association of Coroners, Kansas City, Mo., Aug. 6, 1955.

Research Report on Safety Belts: Crash Injury Research Reports on Safety Belts of 1947 and 1948, National Safety Council, 1952.

Roettig, L. C., *et al.*: Traumatic rupture of the spleen, Am. J. Surg. 2:292, 1943.

Ruff, S.: German Aviation Medicine, World War II, Air Force, Government Printing Office, Washington, D.C., 1950 (out of print).

Severy, D. M.: Automobile Crash Effects, California State Governor's Safety Conference, Sacramento, Oct. 7, 1954.

Teare, D.: Post-mortem examinations in air crash victims, Brit. M. J. 2:707-708, 1951.

Wright, L. T., *et al.*: Traumatic rupture of the liver without penetrating wounds, Arch. Surg. 54:613, 1947.

8

Extremity Injuries: The Common Denominator

Injuries of the extremities are those received most frequently by motorist casualties. Relevant data for this chapter were extracted from 378 patient records, or 57 per cent of a total series of 661 motorist-casualty survivors who had been admitted to the hospital.

The very frequency of injuries of the extremities has resulted in a complacency that is not warranted from the medical and the engineering implications of these injuries.

Although, usually, injuries of the extremities are not in themselves dangerous to life, they may give rise to very serious complications, such as fat and thrombotic embolism, infection, renal failure (in varying degrees) and other peripheral complications such as subfascial hydrostatic compression from bleeding and edema, segmental arterial spasm and nerve damage.

The tendency for persons with severe injuries of the extremities to manifest varying degrees of shock (wound shock) is well known to medical attendants; but it needs to be re-emphasized for lay groups, who must become more aware of the requirements of first-aid dressings and the splinting of obvious fractures. Finally, epidemiologic concepts of trauma in general, and motorist types in particular, are best appreciated from considerations of such fractures as the so-called sideswipe or car-window elbow accidental injuries.

So much for the present regarding the reasons that compel medical interest in injuries of the extremities. What are the implications that should alert engineers and automotive designers to a more objective evaluation of these situations, in conjunction with those of the head and the chest? The answer is not easy. It may be that we have become so engrossed in the study of motorist fatalities that the supplementary answers in the mass of survivors may be escaping our notice? DeHaven must have had this idea in mind when he chose 18 body areas for analysis of airplane survivors.

For example, should there be a specific law of the limits of elasticity for normal bone, the place to look for it would be the long bones. Moreover, the largest and the strongest bone in the body—the femur—is a part of this category. Fractures of it are the most disabling. From the sitting position this bone points forward in the direction of impact, and is accelerated by forward accidental deceleration like an arrow or a missile. Finally, this member of the human mechanical system is at least as close to impact areas as is the chest. More could be learned from studies of the femur in relation to crash forces and their effects.

STATISTICS

Figure 22 establishes the age distribution of this series in decades. All decades through the 9th are represented. There is a sharp rise in the 2nd and the 3rd decades, after which the incidence decline sloped downward steeply. The sexes were about equally divided. Males predominated through the 4th decade; females, afterward. Forty-three

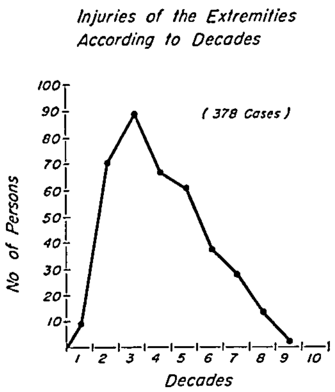


FIG. 22. Graphic representation of incidence of injuries to the extremities in a series of 378 motorist casualty survivors according to decades.

per cent were drivers. Ratio of males to females was 3:1 here. The ratio was almost exactly reversed in regard to the right front seat passengers, and females also predominated in regard to back seat passengers and unclassified categories. Thirty-three per cent of the series were in the front right seat, 6 per cent were in the back seat and 18 per cent were unclassified.

Twenty per cent of this series had injuries of the extremities alone; 80 per cent had concurrent injuries in different parts of the body. In one third of those with extremity injuries alone, more than 1 extremity was involved,

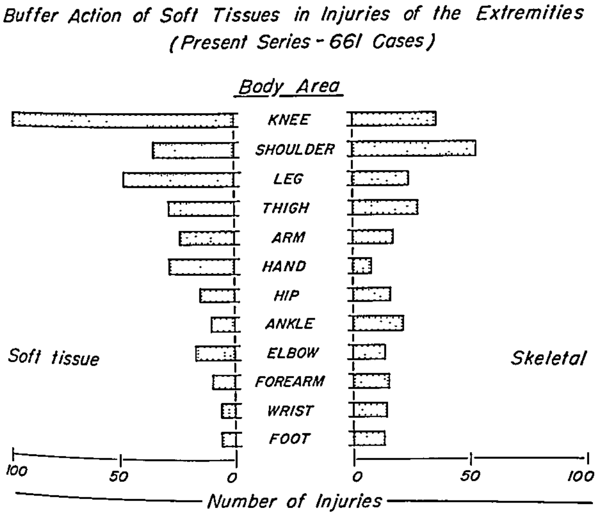


FIG. 23. Graphic representation of incidence and order of rank of soft tissue and fracture injuries of the extremities in a series of 378 motorist casualty survivors.

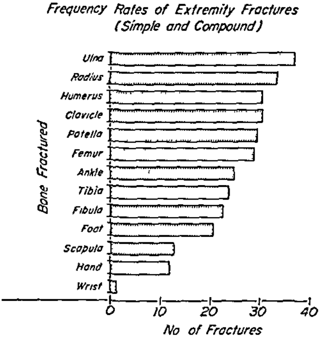


FIG. 24. Graphic representation of frequency rates of fractures (simple and compound) of the extremities in a series of 378 motorist casualty survivors.

however. Moreover, in this lesser group, fractures were twice as frequent as soft tissue injuries. Eight of these fractures were compounded. Upper and lower extremities had about equal numbers of fractures. The prominence of skeletal injuries in this particular subgroup is to be noted, because, for the series of injuries of the extremities as a whole, soft tissue injuries predominated, especially in regard to the lower extremities, where soft tissue lesions outnumbered fractures 3:1.

Figure 23 establishes the order ranks of the different parts of the extremities and also differentiates between soft tissue and skeletal injuries in these regions. The over-all incidence rates in this regard were knee, shoulder, leg, thigh, arm, hand, hip, ankle, elbow, forearm, wrist and foot. There were 579 injuries in all 243 involved the upper extremity; 336, the lower. In the upper extremity, there were 116 soft tissue and 127 skeletal injuries. In the lower extremity, there were 199 soft tissue and 137 skeletal injuries. For the entire series there were 315 soft tissue and 264 skeletal injuries. It should be mentioned that fractures were about evenly divided between the upper and the lower extremities, while soft tissue injuries numbered 199 in the lowers and 116 in the uppers. Approximately 60 per cent of all the injuries of the extremities occurred in the lower ones. Sixty-three per cent of all the soft tissue injuries occurred in the lower extremities. Fifty-two per cent of the skeletal injuries occurred in the lower extremities. Fifty-four per cent of the injuries were soft tissue (chiefly contusive), and 46 per cent were skeletal or fractures.

Figure 23 shows graphically that there are a few areas in which soft tissue lesions out-

TABLE 21. FREQUENCY OF FRACTURES AND DISLOCATIONS OF THE EXTREMITIES IN 378 MOTORIST CASUALTY SURVIVORS

Bone Affected	Simple Fracture	Compound Fracture	Dislocation	Total No.
Clavicle	30	0	6	36
Scapula	12	0	0	12
Humerus	27	3	5	35
Ulna	33	4	0	37
Radius	30	3	0	33
Wrist	1	0	0	1
Hand	9	2	1	12
	142	12	12	166
Femur	26	2	14	42
Patella	26	3	0	29
Tibia	15	8	1	24
Fibula	16	6	0	22
Ankle	21	3	0	24
Foot	18	0	2	20
	122	22	17	161
Total	264	34	29	327

Three humeral fractures and 1 fracture dislocation at the hip were complicated by nerve injuries (radial and sciatic). There was 1 upper humeral slipped epiphysis. There was only 1 medial dislocation of the clavicle (sternoclavicular). (Hip injuries will be discussed in the chapter on the pelvis.) There was 1 tibial dislocation at the knee joint. All humeral dislocations were at the shoulder joint. Hind-foot dislocations included 1 astraguloscaphoid and 1 astragulocalcaneal dislocation.

numbered the fractures, and vice versa. The peak differential in this regard occurred at the knee joint. The discrepancy between soft tissue and skeletal injury in this region may be accounted for by the natural ability of the front of the knee to absorb impacts. In such cases, these were due chiefly to knee-dash contacts. In some instances, occupants becoming aware of an impending crash attempt to brace with their feet and legs, and go down hard on to the floor (in the back seat) or the floor and, rarely, the fire wall (up front) upon the knees.

This sparing action on the bones is canceled out somewhat in the number of femoral fractures and hip injuries that result from such knee impacts. Aside from knee-dash impacts, which occurred more frequently in drivers, it seemed to make little difference where one sat in the car in so far as injuries of the extremities were concerned under crash conditions. A small exception to this generalization derived from the slightly more frequent injuries of the upper extremities and the hands in drivers—probably due to the abnormal leverages and the impacts exerted by the steering controls and by bracing against them.

All the skeletal injuries, i.e., simple and compound fractures and dislocations, as they affected the upper and the lower extremities, were listed (Fig. 24 and Table 21). The 327 lesions were quite evenly divided between uppers and lowers. There was a greater rate of compound fractures in the lower extremities. Due to the number of dislocations of the hip, the lowers predominated in this class of injuries also. All dislocations were single events, while fractures frequently affected 2 or more bones.

Twenty-one of the 29 dislocations occurred in males, and two thirds of them were drivers. The majority of the hip injuries resulted from forward types of collisions. Three humeral and 1 fracture dislocation of the hip were complicated by nerve damage (radial and sciatic). There was 1 upper epiphysial humeral separation. The clavicular dislocations included one medial one (sternoclavicular) in a boy sitting in the right front seat. There was 1 dislocation of the tibia at the knee joint. All humeral dislocations were at the shoulder. Hindfoot dislocations included 1 astraguloscaphoid and 1 astragulocalcaneal lesion.

The 34 compound fractures occurred in 20 persons; 13 were in males, chiefly drivers. In these lesions there were several avulsion types of injuries: 2 to the hand digits and 1 involving an arm that had been looped through a side sling at the time of the accident.

On the whole, fractures occurred with about equal frequency in all occupants of cars, regardless of where they were sitting at the time of accident. More specifically, the frequency rates of fractures in drivers and front seat and back seat passengers were 49, 65 and 68 per cent, respectively; the rate for the unclassified ones was 46 per cent. As already stated, drivers were a little more liable to fractures of the upper extremities.

Despite the common occurrence of fractures of the extremities, in all parts of the vehicles, the relationship between injury and impact areas was quite unmistakable in many instances, and gave distinctive or predictable results. Bracing already has been alluded to. However, several telescoping types of femoral shaft fractures occurred in this manner in elderly back seat passengers from knee-floor contacts. In 1 of these, the fracture was impacted thus, and was allowed to heal this way with some shortening. Several fractures of the small bones of the feet and the ankle resulted from similar causes. Up forward, drivers not infrequently impacted the dash, the steering column, the handle of the emergency brake and door handles, and feet became entangled in foot controls. In the back seat, feet occasionally got caught under the front seat and loosened rugs. Several legs became jammed in partially opened doors. Preimpact positions, such as crossed knees, not infrequently de-

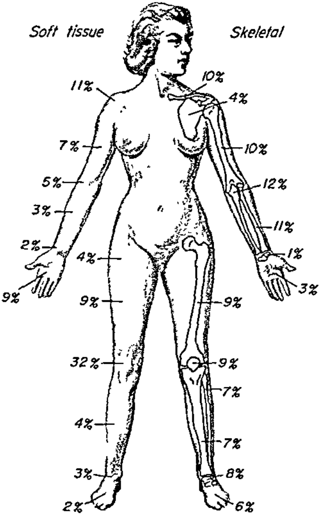


FIG. 25. Pictorial representation of the incidence (rates) of soft tissue and fracture injuries in a series of 378 motorist casualty survivors who received injuries of the extremities.

TABLE 22. NATURE OF INJURIES RELATED TO PRINCIPAL IMPACTS IN REGARD TO INJURIES OF THE EXTREMITIES IN MOTORIST CASUALTY SURVIVORS

PRINCIPAL IMPACTS	NO. OF SOFT TISSUE INJURIES	NO. OF FRACTURES
Forward vehicular collisions	37	42
Ejected from vehicle	22	4
Roll-over	15	8
Ran off road	8	9
Collision from side	5	11
Collision with fixed object	7	8
Lost control	5	6
Rear-end collision	2	1

The great number of vehicular crashes focuses attention upon driver faults. By the same token, the paucity of mechanical failures lauds safety engineering or reliability of the automobile vehicle performance (both mechanical failures were due to rear tire blowouts).

termined the type and the severity of the leg injury. The "window-ledge elbow fracture" will be referred to again.

Figure 25 graphically illustrates the order of frequency of all fractures of the extremities (including the compound ones). From the standpoint of percentages, the clavicle and the humerus are equal, 10 per cent. One is reminded that straps cross the clavicles in the traditional harness worn by airplane pilots The rates of the humerus and the femur are about the same The largest discrepancies exist between the wrist (less than 1%) and the ankle (8%). Some discrepancies exist between forearm and leg, and between hand and foot. The chief injuries of the scapula occurred at the acromion process.

PRINCIPAL IMPACTS

Some relationship was established between principal impacts and the severity of the injuries (Table 22), i.e., soft tissue and fractures.

It is well to point out that there were only 2 mechanical failures in this series (both rear tire blowouts), and that, as usual, the vast majority of accidents involved 2 or more drivers (human elements of accidents). Four train collisions are included in the side impacts. It is surprising to find that forcible ejection caused only 4 fractures, as against 22 soft tissue injuries. In this connection, it should be remembered that serious injuries usually are caused by crushing forces such as result from being run over or pinned under one's own vehicle. Being thrown on to soft ground or snow off the highway frequently causes little or no bodily damage. Be that as it may, there seems to be little connection between type of accident and

type of injury in regard to the extremities. However, more fractures occurred with forward collisions than from any other sources of crash forces. The low incidence of fractures from roll-overs may have been due to the fact that the body was kept well against the seat by centrifugal forces; the same kind that sometimes centrifuge the occupant out on to the highway. Side collisions, in addition to so-called sideswipe elbow, often throw occupants up against projections of the doors and cause fractures.

BIOMECHANICS

Carothers *et al.* reported that 5 whole femurs tested in vertical compression failed under an average maximum load of 1,990 pounds. Four of the failures occurred through the neck of the femur in a manner similar to clinical adduction type fractures. The 5th failure, occurring at 2,390 pounds, was by transverse fracture through the femur shaft (quoted by Snyder).

Evans, Pedersen and Lissner reported failures at different areas of the femur under both static and dynamic loadings. Two tests with dynamic loadings on the greater trochanter resulted in transverse fractures at the neck from 400 and 308 in. pounds. These lower figures and the relatively infrequent neck fractures, as compared with the wide variety of femoral injuries, indicate a need for further study. Evans reviewed these studies (1952) to determine the behavior of the femur as a whole to various stress conditions. It required approximately 3 to 4 times the dynamic loads to produce the same by static loadings.

PROPHYLAXIS

It goes almost without saying that some of the fractures of the extremities were due to seats being loosened and torn from their moorings. Be that as it may, it becomes ever more evident that flailing extremities and severe kinematics of the body in certain types of accidents expose and make these body parts very vulnerable to injuries of all kinds. The problem of prophylaxis is hampered by the fact that the femur, the strongest bone in the body, can be fractured by energy inputs as low as 400-in. pounds dynamically loaded, or about 1,200 in. pounds applied statically. It would seem that protection for extremities will have to be accomplished by indirect means and approaches; i.e., bodily restraint, smooth interiors and increasing distances between extremities and injury potentials. Perhaps changes in driver control design will have a beneficial effect. The principle of force counterbalance has been stated in Chapter 2.

COMMENTS ON TREATMENT

It cannot be repeated too often that the basic responsibility of medical groups in regard to injuries of all kinds is their active and specific treatment. This begins, of course, with first aid. Frequently, the first to render this is a doctor on the scene or at the hospital.

The question of immediate transportation ought to be thought through. Unless the patient is in shock, in so far as primary injuries of the extremities are concerned, he can be transported safely to hospitals where he can get definitive treatment. Naturally this statement refers to the more severe fractures, especially those of the femur, and is not to be construed as a criticism of the many small hospitals and their staffs over the country, without which the mortality in motorist accidents would become even more grisly than is already the case. It is a fact, however, that some family disruptions could be lessened by removing the victim to as close to home as possible. Therefore, whenever possible, he should be removed from the scene of accident to his own community or to a center where his more serious injuries can be attended to The return of motorist victims to their own communities as soon as possible would stimulate more intensive programs of accident prevention, as well as better facilities for their care in their own communities.



FIG. 26. Compound fracture of ankle to which tourniquet had been applied after the man was admitted to the outpatient department of the hospital. Oddly enough, tourniquets rarely are necessary, since reflex arterial spasm usually takes care of that.

OPEN REDUCTION

The treatment of complicated fractures requires the dexterity and the versatility expected of any surgeon plus special training and broad judgment in regard to which of the 3 basic methods of treatment available is to be employed in the case in hand. The question of open versus closed reduction and fixation is a frequent one. In most cases of compound fracture (Fig. 26), the answer is resolved easily. Here it is a matter of experience and following out well-known principles and follow-up care. But in regard to closed fractures, this is not always the case, even from the standpoint of surgical capabilities or the patient's wishes alone. Add to this that the greater the time interval between injury and decision, the greater the hazards and the chance of failure, and one has the whole picture in mind. Medical attendants who undertake the treatment of all kinds of fractures would do well to obtain orthopaedic consultation in the complicated ones and/or transfer these cases to his care. The orthopaedist must distinguish between "operation by virtue of necessity" and "operation as the method of choice" (Clay Ray Murray) in the patients submitted to his care.

SUMMARY AND CONCLUSIONS

Injuries of the extremities are the most common lesions received by motorist casualties. In addition to their importance from the statistical standpoint, they have other important clinical and engineering implications that still call for more adequate consideration and solution. The close relationship between the biomechanical factors in fractures of the extremities and the mechanics of their production—if recognized—would do much to clear up matters of reduction and fixation, especially the indications for open operative methods. Prophylaxis in these areas poses many problems for automotive engineers, and most of them will have to be resolved through indirect methods of protecting occupants.

CASE REPORTS

Case 1 (Fig. 27). Male, aged 47 years. This man was admitted to the hospital on October 17, 1955, 2 weeks after having been ejected forci-

FIG 27 (*Illustration on facing page*). Example of open reduction and internal fixation by necessity. (*Top*) Flank and right lower extremity in adult male 2 weeks after severe forward vehicular collision The patient, who was driving a station wagon, was ejected. Injuries included: multiple contusions, abrasions and lacerations; contusion of the brain, crushed right chest, comminuted fracture of the right femur; and retroperitoneal and mesenteric contusions and hemorrhages. (*Center, left*) Comminuted fragments of femur exposed Also shows severely macerated muscles and partially sequestrated bones. (*Center right*) Shows one of the major fragments (in middle lower third). (*Bottom, left*) Insertion of Streeter intramedullary rod, retrograde. (*Bottom, right*) The rod has been placed completely; the major middle third fragment has been placed over the gap and secured by 2 stainless steel wire loops The latter were overlapped slightly at each end. Bone chips then were added from the right anterior iliac crest. Plaster spica followed H[illegible]ing well advanced May, 1956.



FIGURE 27 (*Caption on facing page*)

bly while driving his station wagon. Details of mechanism unknown.

Diagnosis. Brain contusion, cerebral concussion, crushed right chest, probable contusion right lung, multiple contusions about the body, especially severe at right posterior flank, shock, mild azotemia, dropfoot on left, crushed but not compounded fracture right mid and lower shaft of the femur with marked hemorrhage into the fascial compartment, fracture left mandible and right clavicle.

Skeletal traction was applied to the right lower extremity for 2 weeks, during which time the condition of the patient was precarious. Then an open reduction was done for the femur, an intramedullary pin and bone grafts from the ilium being utilized. The latter were deemed to be essential, since some of the fragments of bone seen at operation already looked to be aseptic. Nevertheless, all fragments, of which there were at least 6 major pieces, were used in the reduction. The soft parts were badly crushed and lacerated. Closure was without drainage, and a single hip spica was applied.

As of November 10, 1955, the patient is doing very well—his general condition is excellent, and all cerebral symptoms have subsided. Prognosis is good for the femur, but healing in these massive injuries usually is quite prolonged.

When last seen (March 31, 1956) healing had progressed to point of full weight-bearing with the aid of a long leg brace. Full recovery of the left drop foot was noted.

Case 2. Female, aged 49 years This patient was admitted to the hospital on September 24, 1955, and was discharged on October 22 with both lower extremities in plaster. She had been a guest passenger in the front seat of a 1954 model sedan being driven by her husband when they became involved in a 5-car collision. Her husband was killed outright; 1 other man died within 48 hours; and she received the following injuries: severe contusion left chest (cardiac contusion suspected but not proved), comminuted fracture right lower radius and ulna, fracture base of proximal phalanx 5th finger of left hand; complete laceration skin and patellar ligament right knee, chip fracture lower pole right patella; fissure fracture left patella, comminuted T fracture of the lower left femur, and lacerations of the [illegible]p and the chin (with mild shock).

The patient was transported 70 miles to the hospital after she had received first aid and sutures of the lacerations. Treatment consisted of repair of the patellar ligament with fascia lata (banked, commercial); open reduction and right angle plating (plus bolting of femoral condyles) of the left femur and cast, closed reduction and cast to right forearm and fixed *skin traction for the finger; together with* medical attention and supervision. Her reactions to injuries and treatment were good. There was considerable dorsal backache and epigastric distress for about 2 weeks, after which these symptoms subsided.

When last seen healing had progressed satisfactorily.

Case 3. Male, aged 31 years. This man was admitted to the hospital on September 3, 1955, and was discharged on September 25. He was a guest passenger in the front seat of a car that was involved in a high speed head-on collision with another vehicle. The driver of the car in which he was riding was injured moderately; the driver of the other car received a fracture of the femur quite similar to his own.

Diagnosis. Multiple contusions, fracture left ankle, comminuted but not compounded fracture of the upper one third of the left femur (up to the greater trochanter), moderate shock, marked subfascial hemorrhage left thigh and secondary anemia.

Treatment consisted of skeletal traction at the lower left femur and the os calcis for 10 days, after which an open reduction was done on the femur, an intramedullary pin and 2 wire loops being utilized. Closure was without drainage, and a spica cast was applied.

The postoperative course has been uneventful.

When last seen healing had progressed satisfactorily.

BIBLIOGRAPHY

Bancroft, F. W., and Marble, H. C.: Surgical Treatment of the Motor-Skeletal System, ed. 2, part 2, Philadelphia, Lippincott, 1951.

Hampton, Oscar P., Jr.: Basic principles in management of open fractures, J.A.M.A. 159:417, 1955.

Kulowski, Jacob: Injuries of the extremities: the most common among motoring casualties, J. South. M. A. 49:165, 1956

Schinz, H R.: Roentgen Diagnosis, New York, Grune & Stratton, 1952.

Watson-Jones, Reginald: Fractures and Joint Injuries, ed. 4, Baltimore, Williams & Wilkins, 1952.

9

Extremity Injuries: Special Considerations

It was not my intention to write a systematic treatise on motorist injuries. For reasons already mentioned, the greatest concern has been with the most common results of motorist accidents. However, certain variants and variables crop up that beg further consideration and elucidation. These are essential digressions. In this chapter will be mentioned some aspects of the epidemiologic implications of motorist injuries, subfascial increased hydraulic pressure and gas bacillus infections.

EPIDEMIOLOGY

The enlarged modern concept of preventive medicine has recently come to include injuries of accidental origin (Gordon). It has been demonstrated that accidents as a health hazard conform in origin and course to the same biologic laws that govern mass disease with specific relationships to time, place and person. In other words, a definite interaction takes place between the agent (vehicle), the host (driver) and the environment (internal and external automotive). Therefore, the causes of origin are very likely to be not a matter of chance for the individual but to rest within the total environment. The basic need is, first, an analysis at the community level and, second, aggressive corrective action at that same level.

Thompson and Chambers felt that the alarming number of "completely unnecessary and extremely serious elbow fractures" (Fig. 28) called for such an analysis. These authors studied 95 patients who had been admitted to Brook's Army Hospital during the 6-year period ending July, 1952, for injuries to elbows that had protruded from car windows under way. In addition, 50 cases already had been cited in the literature. Eighteen of the 95 persons admitted to Brooks had lost an arm above the elbow as a result of car-window accidental injuries. There were 162 separate and distinct fractures. Sixty-three per cent involved the elbow joint proper. Sixty were simple, and 102 were compound fractures. Of the many and varied complications, there were 3 ruptured brachial arteries with subsequent gangrene, 2 of which required amputation through the arm and 1 through the digits. One hundred and forty-four operations were performed on this group; many more electives were contemplated.

In their epidemiologic analysis (admirably done, and one of the few of its kind in the medical literature), special attention was given to the causative principal impacts. Over 80 per cent of these injuries resulted from angular, carom or so-called sideswiping impacts. Here the human element is especially glaring from the standpoint of etiology, because ordinarily these types of impacts are least injurious to occupants and of smaller magnitude due to increased decelerative distances of the vehicles. In other words, many of these impacts are glancing. However, the injury pattern resulting from these injury-accident-impacts is predictable, destructive and utterly uncalled for—therefore, preventable.

There can be no doubt that the vast majority of these injuries are due to the mechanisms described above. However, there are some exceptions to this rule. Some very severe injuries to the elbow regions have





FIG. 28. (*Top*) The child sat between his father, who was driving, and his mother, and received only a slight laceration of the lower lip. The mother was uninjured. The father received sideswipe compound fracture of the left elbow (*bottom*). New car was demolished. This kind of injury is a special case of double jeopardy; i e., exposed to crash dangers of internal and external automotive environments



FIG. 29. An adult female who had impacted the dash from the right front seat in head-on vehicular collision with both knees: compound fractures of the right patella and the left femoral shaft. Her son, who was driving, was not injured.



FIG. 30. Complete rupture of the right popliteal vessels and nerves after 24 hours. Gangrene intervened. Amputation followed. The postoperative course was a mild and modified type of crush syndrome.

been treated by the author as a result of hard right angle (from the side) impacts; from the left for drivers (and left rear seat passengers) and from the right for the right front seat passengers (and right back seat ones) Undoubtedly, also, there are those who are ashamed to admit that their elbows had been sticking out of the window, in view of the campaign that has been waged in educating occupants of automobiles to keep their elbows inside the car.

The preventive measures suggested by Thompson and Chambers included changes in design as follows: window to be made less comfortable;* width of trucks to be limited by general width of present roads and highways; improved highways and roads; mechanical signals instead of arm ones; and,

* Dr. Duncan C McKeever made this suggestion to the writer: simply limit the distance to which the driver's window can be lowered, making it impossible for him to rest his elbow on the ledge



FIG. 31. Teen-age driver with compound sideswipe fracture of the left elbow with subfascial swelling threatening the extremity. Oddly enough, the open wounds did not spontaneously decompress the elbow. This was done by the author's method of multiple puncture drainage. Good recovery (1950).



FIG. 32. Severe fracture of the os calcis complicated by marked subfascial swelling which was controlled by multiple puncture drainage. The patient, who was driving, also had fracture-dislocation of the hip joint on the same side. Good result (1953).

last but far from least, the education of the public (drivers especially) in regard to the real dangers of driving with the elbow resting on the car-window ledge. Tersely stated by Thompson and Chambers, "To retain the elbow in continuity with the brain, the public must contain the elbow within the confines of the vehicles."

There are, of course, other injuries of orthopaedic and general interest that are just as significant from the standpoint of occupant-design relationships—the fracture dislocation or simple dislocation of the hip from knee-dash impacts (Fig. 29) and face, head, chest and abdominal impacts on to steering controls, windshield and dash. But none of these situations bridges, let us say, the internal automotive to the external automotive environments to such a conclusive extent, save in the cases of forcible ejection from the vehicle, as exemplified in the car-window injuries of the elbow. Since almost every medical attendant sees this kind of injury at some time, the realization of its epidemiologic implications should do much to influence medical thinking along these lines.

SUBFASCIAL HYDRAULIC COMPRESSIONS

Three mechanical situations arise, especially from severe injuries due to impacts that often cause pressures that may threaten the integrity of the part affected. These situations are due chiefly to increased hydraulic subfascial pressure caused by bleeding (Fig. 30), exudates and intrinsic tissue swelling or reaction, and hydrarthrosis or hemarthrosis.

The dangers attending impending local circulatory failure have long been appreciated by layman and doctor alike, especially in regard to supracondylar fractures of the elbow in children. Not so well appreciated, perhaps, are the not infrequent similar situations that result elsewhere in the extremities after fractures and/or contusive injuries of the soft parts. Most of those observed by me have resulted from motorist impacts, and

have appeared as subfascial increased pressures from bleeding, edema or other tissue changes of unknown origin (?) that involve the muscles chiefly. Such painful swellings, which have threatened the integrity of extremities or portions affected, occur most commonly at the elbow (Fig. 31) and the forearm, but they have also developed in the wrist and the hand, and in lateral and other compartments of the leg below the knee; in the plantar spaces; and also, less commonly, on the dorsum of the foot (Fig. 32). In some of these cases, there were associated severe injuries proximal to the peripheral lesions under issue, which brings up the question of the role of segmental arterial spasm in these injuries, in addition to the local peripheral damage. In most of my examples, the local reaction was purely that due to the local injury. The picture clinically in these cases resembled closely that of impending Volkmann's paralysis.

In my experience with this disturbing condition, I found it very efficacious to relieve the subfascial spaces under tension by incisions made under full operative exposure, followed by loose closure of the skin with or without drainage in accordance with the indications of the individual case. This has been recommended for elbow lesions, in addition to other direct and indirect approaches toward improving the circulatory embarrassment, and it should be watched for especially after surgical procedures upon the more severely injured extremities.

In the past 8 or 9 years (method unpublished previously), I have employed a faster and a more simple method of draining or decompressing the subfascial spaces that obviously are under dangerous hydrostatic stress This consists of making a large number of punctate stabbing wounds through the skin and the subcutaneous tissue (and through the fascia as well) over the entire affected area, close together and under sterile precautions In a series of at least 25 cases, there was only 1 infection (in that of a severe elbow fracture in an adult who had been subjected to operative treatment, a person who was allergic to every known antibiotic and other essential drug). The patient recovered, with a good functional result in spite of the stormy postoperative course. The procedure of multiple punctate decompression should not be withheld after the subfascial pressure has developed to the extent that painful induration has become obvious. Petrolatum dressings and cast or splinting as indicated usually follow. It goes almost without saying that every precaution is taken during punctate drainage to avoid the larger arteries and veins.

Of course, the special situation in regard to subfascial hydrostatic stress always will remain the anterior part of the elbow joint in supracondylar fractures of children. For this situation, in the face of impending Volkmann's contracture, I learned recently that injection of procaine solution (1%) directly into the medial nerve at the site of injury will relieve reflex segmental arterial spasm (after exposure and open reduction of the fragments).

Two other points in connection with these fractures are worth mentioning. First in my opinion, a considerable amount of the immediate swelling in these fractures is due to bleeding into the joint (Fig. 33). This portion of the over-all swelling is relieved simply by joint aspiration. Second, in my experience, this fracture is one definite exception to the rule of immediate reduction and fixation. I have found that reduction is accomplished just as easily some 12 to 24 hours afterward, and immobilization is effected far better then (by posterior splint only—never a circular cast) than when it is done right away. In other words, a reasonable period of recumbency and elevation of the arm on a pillow is good for both patient and doctor.

GAS BACILLUS INFECTION

The emergency care of injuries of the extremities has been discussed often and fully everywhere. I wish to stress one point in regard to possible gas bacillus complications in



FIG. 33. Self-decompressive fracture of right ankle (shoe and nylon ripped off) received by adult woman driver in high speed head-on collision with another vehicle. Also received fracture of the sternum from impacting steering controls. She recovered. Her mother, who was sitting next to her, received severe fracture of the left femur, shock and multiple topical lesions. She also recovered.



FIG. [illegible] bacillus [illegible] fracture [illegible] young man with crushing injury. High amputation was successful in saving this man's life. (*Top*) Lateral side of leg. (*Bottom*) Medial side of leg.

compound fractures and in some severely lacerated soft tissue injuries. From the standpoint of diagnosis, it should be remembered that this complication occurs in a matter of a few hours. It can be detected readily by the experienced roentgenologist when the patient is admitted to the hospital, as he will differentiate gas bacillus from air that merely has been sucked into the wound at the time of the accident. Therefore, roentgenograms should be taken of these injured areas immediately after the patient is admitted to the emergency room, with the possibility of gas infection in mind, and transmitted to the roentgenologist. Moreover, I make it a practice to send cultures and pieces of tissue to the laboratory for gas bacillus investigation in all cases that suggest its possibility. Early diagnoses in these dangerous infections often spell the difference between saving a limb or amputation; or it may even be the difference between life and death. The hazards of these complications of compound fractures go far beyond those reflected in their relative infrequency in motorist-casualty survivors.

In accident, the specter of gas bacillus infection always hovers over open fractures (Fig. 34), especially those with severe soft tissue involvement or comminution of the bone fragments.

Some years ago, I had under my observation in the hospital at the same time 7 motorists with compound fractures that had been complicated by gas bacillus infections and osteomyelitis. All but one of these had to undergo 1 or more amputations. Gas bacillus complications still occur from highway accidents, despite improved technics in surgery and the liberal use of antibiotics, in conjunction with antitetanic and antigas bacillus antitoxins and serums. Less than a year ago I treated a severe gas bacillus infection in a compound fracture of the femur in a young motorcycle casualty. The leg was saved by the miracle of modern therapeutic modalities, including x-ray.

On the other hand, several months ago a gas bacillus infection complicated a severely crushed compound fracture of the femur in which primary amputation had been done when the patient was admitted to the hospital. Within 48 hours, a hip high revision (guillotine) had to be done that was fol-

lowed by months of repeated drainages, during which time the patient received almost 20 pints of blood.

In November, 1955, I did a guillotine midthigh amputation on a man who had received a severely crushed compound fracture of the right ankle and crushed simple fracture of the lower right femur. The injuries were received from a power part of a cornpicker. I saw the man 30 hours after injury, when gas was detectable by roentgenogram and clinically to the knee. I decided to amputate, but was overruled by several who wished to try conservative treatment after débridement. Débridement was thorough, as was the rest of the systemic and local management. The next day the man was deathly sick, and the gas was above the knee. He made a prompt recovery after amputation.

This much can be said for established gas bacillus infection of the *Bacillus welchii* type. There are no variants to puzzle the clinician; all are alike in their deadliness and appearance and good response to prompt amputation.

SUMMARY AND CONCLUSIONS

It so happens that the 3 subjects for special mention in this chapter are closely interrelated; i.e., severe elbow injuries frequently embody all of them—such as untoward swelling and liability to infections of all kinds.

Voluntary observance of the common sense practice of keeping the arm within the car will eliminate the sideswipe types of the severe elbow injuries under discussion.

The elbow fractures present challenging situations, and often pose questions regarding the integrity of the arm from 3 angles of approach: (1) joint effusion; (2) subfascial pressure; and (3) reflex arterial segmental spasm

Therapeutic suggestions have been offered to combat these 3 apocalyptic complications.

The fourth is gas bacillus infection. Antibiotics have subdued but not yet conquered this deadly threat to life and limb.

CASE REPORTS

Case 1. Male, aged 55 years. Was admitted to the hospital on March 26, 1955, and was discharged on July 1, 1955.

Final Diagnosis. (1) Primary shock; (2) crushed right chest; (3) subcutaneous emphysema of right axilla; (4) multiple contusions and abrasions of face and extremities (old amputated right hand); (5) fractures of shaft of left femur; (6) fracture of acromion process, right scapula; (7) fracture of medial end of right clavicle; (8) compound comminuted fracture of right lower and shaft of femur; (9) partial traumatic amputation of right thigh with marked tearing and crushing of all the muscles and the skin except for a portion of the medial hamstrings and adductors, with gross contamination of the lacerated parts; (10) gas bacillus infection of right thigh.

The above-mentioned injuries were received as a front seat passenger in one of the heavier models (1952) sedan that collided head on at high speed with a trailer truck. The driver of the sedan was killed instantaneously. The patient was jammed forward between partially opened door and door frame. The sedan was demolished. The truck partially left the highway and turned about 90°. The driver of the truck was not hurt. Amputation at the right thigh was done 2 hours after the patient was admitted to surgery, at which time his blood pressure had been raised from 90/60 to 120 70. A few skin sutures were taken. Several rubber tissue drains were placed, as well as a catheter, for penicillin solution. He was given the usual prophylactic doses of antitetanic and antigas bacillus serum, along with penicillin and streptomycin intramuscularly.

On the following day the chief concern was a marked paralytic ileus and threat of serious damage to the right lung. In the meantime, the laboratory reported cultures of oganisms (from the operative field) that were grown anaerobically; nonmobile spore forming; and, on the following day these were reported to be definitely gas formers of the *Clostridium welchii* type. But at this time, 36 hours after admission, there was no need for laboratory confirmation of gas bacillus infection, because both the odor and the appearance of the amputation stump were typical of this dreaded complication. A guillotine revision of the stump just below the greater trochanter was done immediately under Pentothal Sodium anesthesia.

Ten days later an intramedullary pin was inserted into the left femur. Several soft tissue abscesses did develop and require drainage. Re-

lowed by months of repeated drainages, during which time the patient received almost 20 pints of blood.

In November, 1955, I did a guillotine midthigh amputation on a man who had received a severely crushed compound fracture of the right ankle and crushed simple fracture of the lower right femur. The injuries were received from a power part of a cornpicker. I saw the man 30 hours after injury, when gas was detectable by roentgenogram and clinically to the knee. I decided to amputate, but was overruled by several who wished to try conservative treatment after débridement. Débridement was thorough, as was the rest of the systemic and local management. The next day the man was deathly sick, and the gas was above the knee. He made a prompt recovery after amputation.

This much can be said for established gas bacillus infection of the *Bacillus welchii* type. There are no variants to puzzle the clinician; all are alike in their deadliness and appearance and good response to prompt amputation.

SUMMARY AND CONCLUSIONS

It so happens that the 3 subjects for special mention in this chapter are closely interrelated; i.e., severe elbow injuries frequently embody all of them—such as untoward swelling and liability to infections of all kinds.

Voluntary observance of the common sense practice of keeping the arm within the car will eliminate the sideswipe types of the severe elbow injuries under discussion.

The elbow fractures present challenging situations, and often pose questions regarding the integrity of the arm from 3 angles of approach: (1) joint effusion; (2) subfascial pressure; and (3) reflex arterial segmental spasm

Therapeutic suggestions have been offered to combat these 3 apocalyptic complications

The fourth is gas bacillus infection. Antibiotics have subdued but not yet conquered this deadly threat to life and limb.

CASE REPORTS

Case I. Male, aged 55 years. Was admitted to the hospital on March 26, 1955, and was discharged on July 1, 1955.

Final Diagnosis. (1) Primary shock; (2) crushed right chest; (3) subcutaneous emphysema of right axilla; (4) multiple contusions and abrasions of face and extremities (old amputated right hand); (5) fractures of shaft of left femur; (6) fracture of acromion process, right scapula; (7) fracture of medial end of right clavicle; (8) compound comminuted fracture of right lower and shaft of femur; (9) partial traumatic amputation of right thigh with marked tearing and crushing of all the muscles and the skin except for a portion of the medial hamstrings and adductors, with gross contamination of the lacerated parts; (10) gas bacillus infection of right thigh.

The above-mentioned injuries were received as a front seat passenger in one of the heavier models (1952) sedan that collided head on at high speed with a trailer truck. The driver of the sedan was killed instantaneously. The patient was jammed forward between partially opened door and door frame. The sedan was demolished. The truck partially left the highway and turned about 90°. The driver of the truck was not hurt. Amputation at the right thigh was done 2 hours after the patient was admitted to surgery, at which time his blood pressure had been raised from 90/60 to 120/70. A few skin sutures were taken. Several rubber tissue drains were placed, as well as a catheter, for penicillin solution. He was given the usual prophylactic doses of antitetanic and antigas bacillus serum, along with penicillin and streptomycin intramuscularly.

On the following day the chief concern was a marked paralytic ileus and threat of serious damage to the right lung. In the meantime, the laboratory reported cultures of oganisms (from the operative field) that were grown anaerobically; nonmobile spore forming; and, on the following day these were reported to be definitely gas formers of the *Clostridium welchii* type. But at this time, 36 hours after admission, there was no need for laboratory confirmation of gas bacillus infection, because both the odor and the appearance of the amputation stump were typical of this dreaded complication. A guillotine revision of the stump just below the greater trochanter was done immediately under Pentothal Sodium anesthesia.

Ten days later an intramedullary pin was inserted into the left femur Several soft tissue abscesses did develop and require drainage. Re-

garding the right stump, this required numerous revisions, drainages and skin grafting.

During his stay in the hospital (an unusually long one even with his numerous injuries and complications), the patient was given 16 pints of blood, several pints of plasma and innumerable bottles of the various nutritive and electrolyte solutions. He became insensitive (that is, his organisms did) to all the antibiotics. He never showed signs of kidney impairment. His chest cleared up spontaneously, the left femur healed, his temperature was normal during his last 2 weeks in the hospital, and his final blood count was quite acceptable.

Case 2. Male, aged 31 years. Was admitted to the hospital on March 22, 1953, and was discharged on April 4.

Final Diagnosis. Severe laceration of forehead; fissure fracture (?) of skull, occipital; posterior fracture dislocation of right hip; fracture of left 4th rib; comminuted fracture of right os calcis; severe subfascial hydraulic pressure of foot and ankle, right; impending ischemia of right foot; mild shock; and secondary anemia.

Subsequent Diagnosis. Aseptic necrosis of right femoral head; mild osteoarthritis of right hip joint; post-traumatic arthritis of right subastragaloid joint.

Orthopaedic Treatment. (1) 1 day after admission a punctate multiple subfascial drainage had to be done and was followed by marked relief of pain and subsidence of ischemia (see Fig. 32) (no attempt was made to improve the position of the heel bone)—however, a cast was applied immediately following the drainage; (2) on the 3rd day after admission, through a Smith-Petersen approach, an open reduction and internal fixation (screw for acetabular rim) was done, with application of spica cast.

Postoperative recovery was good. Subsequent aseptic necrosis of the femoral head is improving under conservative treatment and guarded weight-bearing.

Case 3. Male, aged 36 years Was admitted to the hospital on March 28, 1950, and was discharged 1 week later. While driving on night of March 27 with arm on window ledge, he was sideswiped by a truck traveling in the opposite direction and received a severe injury of the elbow. He was given first-aid treatment and sent to the hospital the following day. His badly comminuted fracture of all the bones of the elbow was débrided and put up on an abduction splint with drainage; despite which he developed a severe infection (no gas bacillus). Several weeks later, a massive sequestrum involving the entire lower one quarter of the humerus had to be removed. Some months later, when the wound had healed, the gap was bridged with a massive tibial graft, which took and healed. Final result was a stiff elbow in a little better than a right angle position, with good function of the hand.

Case 4. Female, 8 years of age. Was admitted to the hospital on April 22, 1955, with a badly comminuted compound supracondylar fracture of the left elbow which she suffered when she fell from a horse. The radial pulse was not palpable; this was thought to be due to excessive swelling and pressure from the bony fragments. Reduction was done under direct vision, but the pulse failed to respond. Brachial artery was exposed and found to be stringlike in consistency without pulsation or other evidences of injury. The median nerve then was injected with 1 per cent procaine hydrochloride solution, and the pulse returned dramatically. Reduction was held with posterior splint. There was active motion in 2 weeks.

The final result was satisfactory without evidence of functional impairment of hand or wrist.

Case 5. Male, 14 years old (said not to have been driving). Was admitted one night in 1952 with a compound fracture of the left elbow after a carom collision with a car traveling in the opposite direction. Despite the fact of spontaneous decompression due to open wound, there were excessive swelling and loss of the radial pulse. The wound was débrided, and additional multiple punctate subfascial drainage also was done, with excellent functional results.

BIBLIOGRAPHY

Console, A. Dale: Segmental arterial spasm associated with supracondylar fracture of the elbow, S. Clin. North America 28:467-472, 1948.

Gordon, John E.: Accident prevention through epidemiologic analysis, J. Michigan M. Soc. 50:1239, 1951.

Kulowski, Jacob: Segmental arterial spasm of the brachial artery; report of case treated by procaine into median nerve, Surgery 38:1087-1089, 1955.

Meleney, Frank L.: Treatise on Surgical Infections, New York, Oxford, 1948.

Selye, H.: Stress, Montreal, Acta Endocrinologica, Inc., 1949.

Thompson, M. S., and Chambers, G. H.: Epidemiology of car window accidents, South. M. J. 46:979, 1951.

10

Crush Syndrome

Lower Nephron Nephrosis

Nowhere, perhaps, is encroachment upon the health of the individual by trauma so dramatically illustrated as in the so-called lower nephron nephrosis.

Lower nephron nephrosis is a disease in which there are degeneration and necrosis of the epithelial cells lining the distal convoluted tubules of the kidneys and the presence of haem casts in the tubules. This results in acute renal failure, which frequently is fatal. This lesion often is referred to as the crush syndrome, because it occurs sometimes after crushing injury to muscle. Although the renal changes were described by Hackradt in 1917, Bywaters and Beal (1941) were the first to recognize the importance of these lesions in the production of traumatic anuria during the bombings of London in 1940. These authors had noted renal failure and death from uremia on or about the 7th day in some patients who had been pinned under fallen debris for variable periods of time. Lucke, in 1946, reported the incidence of this disease in battle casualties, and described the pathologic process in detail. Moreover, he stressed the reversibility of the lesion, and called attention to many other conditions that are characterized by similar renal pathology. A lower nephron nephrosis may occur, in addition to crushing injuries of the muscles, in nontraumatic muscular ischemia, burns, transfusion with incompatible blood, heat stroke, blackwater fever, toxemia of pregnancy, alkalosis and sulfonamide intoxication, and following transurethral prostatic resection. This subject, therefore, is of special interest in relation to injuries of the extremities and to general problems of extrarenal azotemia and renal disease.

PATHOGENESIS

One view is that the haem casts block the tubules, causing a mechanical obstruction; the other, that in the presence of tubular necrosis, massive reabsorption of the glomerular filtrate is unselective through the nonfunctioning tubule, which now acts only as a semipermeable membrane. Be that as it may, renal failure ensues. The course of the disease is relatively brief, usually from 3 to 10 days; and the tendency is for spontaneous recovery. The 2 conditions that may give rise to this condition and concern us in this chapter are crushing injuries and nontraumatic muscular ischemia. Bywaters and Dible reported that the latter occurred in 2 cases of injury in automobile accidents, involving ischemia of large muscle masses. For the former, there follow some shock, passage of smoky urine (giving a positive Benzedrine reaction, due to myohemoglobin, oliguria or anuria with mounting blood nonnitrogen protein). Recovery will follow if urinary output is re-established. Six more or less typical features are involved in the diagnosis: (1) type of injury; (2) first phase of shock; (3) *second phase of shock*; (4) changes in the urine; (5) changes in the blood; and (6) local tissue damage.

Clinically, aside from his coincidental injuries, the victim of a crushing injury appears to be quite well. With swelling due to loss of plasma into the injured limb, shock

ensues (due to damaged capillaries). There is hemoconcentration, but the blood pressure is about normal so long as peripheral compensatory vasoconstriction continues. When this mechanism fails, the blood pressure falls rapidly. With adequate treatment, there is a favorable response until the first passage of urine, which is pathologic, falls in output and is associated with a rise in blood pressure to pathologic levels. The blood urea, nonprotein nitrogen, potassium and phosphate rise also, while the carbon dioxide combining power of the plasma falls. A sudden diuresis on or about the 7th day indicates a favorable response and prognosis.

Meanwhile, the damaged limb swells further and becomes quite firm. Blistering follows (Fig. 35), and there are signs of impending circulatory failure, persistence of which may presage gangrene. These changes will be noted on microscopic examination. Oliguria and anuria are attention-arresting phenomena that hold too alarming a place in the mind of the surgeon. If the toxins are eliminated, normal circulation is restored and the life of the patient is preserved long enough, the kidney tubules will regenerate and resume their normal function. It should be remembered that the greatest danger in the early stages is not from the byproducts of metabolism but from retention of fluids and imbalance of electrolytes. The entire electrolyte balance, not any particular one, is upset.

The treatment advocated is undertaken in somewhat the order of that adhered to in previous chapters; that is, the prophylaxis comes first, followed by local treatment and attention to the renal condition. Preventive measures are to be undertaken at once by rescue parties attending to burial type of body stresses, and include alkalinization of the urine and sedation for shock. The local tissue damage and/or response to supportive measures will determine the surgical technics and policies for the individual case. Amputation may be indicated, and should not be delayed too long once such decision is made



FIG. 35. Telltale signs of subfascial increased hydrostatic pressure (toxic ?) and/or reflex arterial segmental spasm: pain, swelling, discoloration and blistering. Simple fracture of tibia and fibula; no treatment for several days. Watch for impaired renal function!

Doubtless, the principles of treatment of the renal and the extrarenal factors demand competent medical (internist) attention. Surgeons never should assume responsibility for the medical treatment of these complex and seriously ill patients. According to Snapper, water balance and maintenance of caloric requirements are the most essential features in this regard. In severe cases it may be necessary to explore extrarenal means to relieve the uremia (artificial kidney, dialysis).

For clinicians who assume responsibility in the treatment of injuries of the extremities, it is well to remember that classic signs and symptoms seldom will occur in any but the more extensively crushed limbs. But, undoubtedly, subclinical levels must be very common, and will be overlooked unless the condition is kept in mind. Above all, prophylactic steps taken early in cases in which the syndrome may be expected to develop will abort most of them, especially in some postoperative patients in whom rather extensive procedures had to be undertaken. All cases with oliguria should be suspect and treated accordingly.

Most recently I had under my care a man who had been buried up to his chin by a cave-in for several hours. He suffered a fracture of the left clavicle and mandible, and complained of bruising of the chest "inside" All baseline laboratory values were estab-



FIG. 36. Typical case of crush syndrome several days after admission to hospital after about 12 hours of exposure to cold and pressure of human body on top of her. No fractures. Recovery was complete and lasting.

lished on the day of his admission to the hospital, and other prophylactic measures were instituted at once. Nothing happened to retard the man's recovery, which was uneventful. Several other casualties were treated similarly with good results. Two exceptions come to mind: in one, the condition of crush syndrome was under anticipated observation; in the other, it was not suspected until afterward, when the patient had recovered.

The first patient who made me aware of this condition in civilian practice was seen late in 1949, 3 days after a light tractor had rolled on to him The young man had an obviously dislocated right knee joint with evidences of rupture of the popliteal artery and vein and established gangrene. Amputation several hours after admission to the hospital confirmed the diagnosis. There was no shock after operation. However, subsequently there was a course of irregular fever and the development of a severe anemia that required 5,000 cc. of whole blood in divided doses over a period of 8 days postoperatively. He recovered without ever having manifested frank symptoms of oliguria. But *it should be pointed out that severe secondary anemia almost always presents a major problem in lower nephron nephrosis.*

Shortly after that, another young man was admitted to the hospital after a small tractor had fallen on him and pinned him down in a puddle of cold water (December) for about 40 minutes. He had fractures of the right tibia and fibula, the pelvis and several lumbar transverse processes. There was contusion of the right flank, but no shock, on admission. Despite adequate precautions, his urinary output dropped to less than 400 cc. for 4 successive days afterward, but returned to 1,000 cc. on a restricted intake. In this case, also, aside from a sudden drop in hemoglobin and red blood cell values to half their normal value on the 2nd day after admission, all other laboratory and clinical investigations for lower nephron nephrosis were within normal limits. This was not the case in the classic example of the disease who was admitted shortly after this one (man recovered uneventfully).

Pauline F., white, single and 30 years of age, was pinned under 2 other occupants inside an overturned car for 11 hours in subzero weather (January 5-6, 1951). When extricated, 1 person was dead. The second, a sister of Pauline, showed extreme shock, mild frostbite of the feet (the shoes on both women had been ripped away during the run off the road and the roll-over), fracture of the 3rd lumbar vertebra, of the right wrist and of almost all the ribs on the left side This woman recovered after a stormy course that included pneumonia, slight azotemia and mild urinary suppression. On the other hand, Pauline presented major medical problems from the very beginning of her admission to the hospital (Fig. 36).

She was in extreme shock for over 12 hours, and her temperature was subnormal during this period. *Both feet and legs to the mid-thighs*

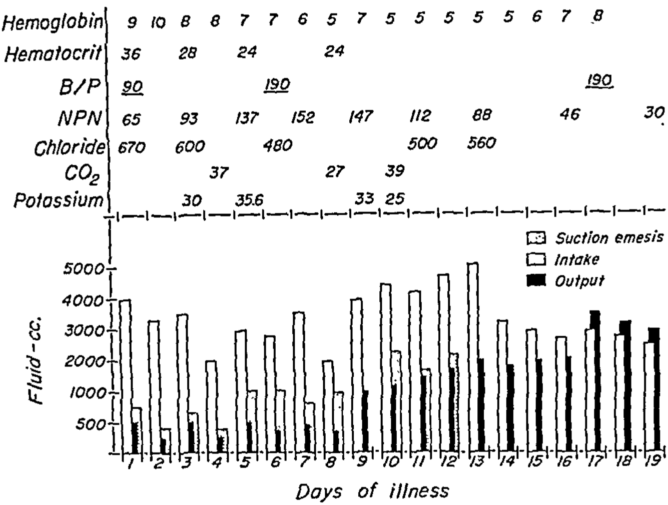


FIG. 37. Graphic representation of blood chemical findings and fluid balance record in case of so-called lower nephron nephrosis. It is interesting to note that high amounts of fluid intake took place despite attempts to keep it down—a danger always to be avoided whenever more than 1 medical attendant participates in the care of these complicated cases. However, recovery was complete.

were frozen severely, pulseless and blue. Her blood pressure rose after the 2nd day to between 170 and 190 systolic. Blood values rose rapidly, and were associated with oliguria. She was given intravenous procaine solutions and cortisone, and was heparinized. On the 2nd day, the legs were swollen and blistered. A day or two later, however, the pulsations began to return. She vomited and retched from the beginning of treatment. Fluid balance was difficult to maintain. Most difficult was the maintenance of caloric needs; the patient lost weight rapidly. On the morning of the 8th hospital day she suddenly developed signs and symptoms of a severe pulmonary edema, which was combated successfully by positive oxygen pressure exerted by an anesthetic machine (thus driving the fluid out of the alveoli into the circulation). Throughout she had a severe secondary anemia. Transfusions were kept to a minimum, because of fear of thromboses in the legs (emboli) and a too rapid return of toxins into the blood stream. Then, quite suddenly on the 8th day of hospitalization, she developed a diuresis (Fig. 37) and slow but definite recovery. She was discharged from the hospital some 21 days after her admission with no loss of tissue from the feet or the legs (which had been treated most conservatively and observed by mild light cradle and cotton protection from undue pressures). She made an excellent recovery without any disability whatsoever.

GENERAL SUMMARY—INJURIES OF EXTREMITIES

Fifty-seven per cent of 661 motorist-casualty survivors manifested injuries of the extremities. Except for the combined in-

juries of the face and the head, traumata to legs and arms rank highest in regard to the different body areas injured in vehicular accidents. Eighty per cent of these casualties had injuries in addition to those of the arms and the legs. Over 40 per cent of these people were drivers. Predictable patterns of injury in regard to extremities are focused on those of the femur, whose relationships between structure and injury were unmistakable. Soft tissue lesions predominated, chiefly in the lowers. Simple fractures far outnumbered the compound ones or dislocations. The ulna was the bone affected most frequently. Certain areas, especially the knee, showed discrepancies between soft tissue and skeletal injury, probably owing to the natural tolerances of these regions to impacts.

Car-window fractures, so called, actually connect the internal and the external automotive environments. Since this is a purely voluntary phenomenon, these injuries have helped to influence medical thinking along modern lines of preventive medical practice in regard to accidental injuries in general and motorist casualties in particular. Thus, the full impact of epidemiologic concepts become evident.

Injuries of the extremities have far-reaching implications in regard to first aid, emergency care and definitive treatment, some of it on a sociopsychological basis.

Several of the most hazardous complications have been discussed; i.e. subfascial pressure, reflex segmental arterial spasm, gas bacillus infection and lower nephron nephrosis.

The injuries of the lower and the upper extremities become the common denominators of motorist injuries from the standpoint of both the medical and the engineering professions. Will these injuries furnish the key to crash-impact prophylaxis?

BIBLIOGRAPHY

Bywaters, E. G., and Dible, J. H.: The renal lesion in traumatic anuria, J. Path & Bact. 56:111-120, 1942.

Gamble, J. L.: Chemical Anatomy, Physiology and Pathology of Extracellular Fluid, Cambridge, Mass., Harvard, 1949.

Kulowski, Jacob: Crush Syndrome, Section B., Proceedings and Seminar Notes of the Orthopaedic Department of Mercy Hospital (Dr. A. Steindler & Staff) 1:2, 1950-1951.

Lucke, B.: Lower nephron nephrosis, Mil. Surgeon 99:371, 1946.

Maclean, John T.: Acute Renal Failure, Springfield, Ill., Thomas, 1952.

Robinson, Cecil: Proceedings and Seminar Notes of the Orthopaedic Department of the Mercy Hospital (Dr. A. Steindler & Staff) 1:2, 1950-1951.

Trueta, J., *et al.*: Studies of Renal Circulation, Springfield, Ill., Thomas, 1947.

11

Acute Motorist Injuries of the Spine

ORIENTATION: NECK, BACK AND PELVIS

The sitting position of automobile occupants stresses the close relationship of neck, back and pelvis. For this reason, Tables 23 and 24 establish the general statistical evaluation of these body areas. Henceforth, this chapter will deal with acute injuries of the cervical and dorsal spine; the following chapter, with the acute motorist injuries of the lumbar spine and the pelvis.

One hundred and eighty-nine, or 28.6 per cent, of 661 motorist casualty survivors received injuries to the neck, the back and the pelvis. This grouping has clinical, as well as crash-impact, implications, especially from the seated position of vehicular occupancy. From this standpoint, the largest body mass is in a state of relative balance. Abrupt vehicular impacts easily upset this balance, the basic manifestation of which is dislocation of the occupant. Injury results from the haphazard bodily impacts that follow upon such dislocations. Thus simply is the basic lesson of safety under crash conditions demonstrated; i.e., maintenance of the body in the seat.

According to seating, the distribution was: drivers, 97 (51.3%); right front seat passengers, 49 (26%); back seat passengers, 11 (5.8%); and unclassified occupants, 32 (16.9%).

Table 23 establishes the general rates and the distribution of the injuries to the different areas of the body under discussion. There were 211 injuries, distributed as follows: neck, 60 (28.4%); back, dorsal, 35 (16.6%); back, lumbar, 61 (28.9%); and pelvis, 55 (26.1%). Injuries to the neck and the lumbar and the pelvic areas were about evenly distributed. The lower incidence of *dorsal injuries was accounted for* probably by the fact that the chest had absorbed so many impact energies.

The higher rates of soft tissue injuries in the cervical and the lumbar regions probably are accounted for by the tendency of these areas to be subjected to inertial stresses. The converse was noted in the pelvic area; i.e., *skeletal injuries outnumbered the soft tissue* ones. This is due partly to the fact that the hip regions suffered considerable forces transmitted through the femurs. In other words, here, too, both direct and indirect forces were operative, but with different results. In the neck and the lumbar areas, sprains were notable, while in the pelvic region, due to the hip injuries, there were more actual skeletal injuries than soft tissue ones.

Table 24 reviews the relationship between the principal impacts and the nature of the injuries received. The high incidence of forward vehicular impacts is to be noted. However, there appears to be a higher ratio of forcible ejections, roll-overs, rear-end collisions and occupant dislocations. These are related to the incidence of sprains and skeletal lesions. Moreover, the almost equal proportions of soft tissue injuries (including sprains) and skeletal injuries (including dislocations and joint injuries) strongly suggest the relationship of sprains and fractures to lesser and greater magnitudes of force, respectively. In other words, increasing order of forces produces an increasing order of lesions with topical ones at one extreme and

TABLE 23. DISTRIBUTION AND FREQUENCY RATES OF SOFT TISSUE AND SKELETAL INJURIES IN 189 MOTORIST CASUALTY SURVIVORS WITH NECK, BACK AND PELVIC LESIONS

BODY AREA AFFECTED	SOFT TISSUE INJURY* No.	%	SKELETAL INJURY† No.	%
Neck....	38	33.6	22	22.5
Dorsum..	20	17.7	15	15.3
Lumbar.	39	34.5	22	22.5
Pelvis....	16	14.20	39	39 7
	113	100.00	98	100.00

* Includes abrasions, contusions, lacerations and sprains
† Includes subluxations, dislocations and fractures

TABLE 24. RELATION OF PRINCIPAL IMPACTS TO NATURE OF INJURIES IN NECK, BACK AND PELVIS

PRINCIPAL IMPACTS	TOTAL NO.	SOFT TISSUE* INJURIES	FRACTURE	SUBLUXATION AND DISLOCATION
Forward vehicular collisions	43	20	18	5
Roll-over	16	9	4	3
Ran off road.	11	4	5	2
Collision with fixed object	9	1	5	3
Rear-end collision.	10	8	0	2
Driver dislocated	8	2	2	4
Forcibly ejected	21	13	8	0
Collision with train	6	3	2	1
Struck from side	5	4	1	0
Lost control	2	1	0	1
	131	65	45	21

* Soft tissue injuries include both topical injuries and sprains

TABLE 25. INCIDENCE OF SOFT TISSUE AND SKELETAL INJURIES OF THE NECK

SOFT TISSUE INJURIES	No.	SKELETAL INJURIES	No.
Contusion	12	Subluxation	10*
Abrasion	3	Dislocation	2
Laceration	2	Fracture: body	5
Sprain.	23	process	5
	40		22

* As used here, *subluxation* means incomplete dislocation, usually rotatory

fractures at the other; with sprains, subluxations, dislocations and fracture dislocations in between.

ACUTE INJURIES OF THE NECK

Nine, or 15 per cent, of those with neck injuries were not complicated by additional injuries elsewhere in the body; the rest were. Table 25 shows the variety and the rates of the different types of injury. There were twice as many soft tissue lesions as skeletal. The frequency of contusions and sprains contrasts with the paucity of lacerations and abrasions. Sprains, which are a type of skel-

etal injury, equal the total number of subluxations, dislocations and fractures.

Due to the anatomic arrangement of the veins, contusive lesions usually localized to the sides of the neck were sometimes much more severe than such injuries in other parts of the body. The dangers from lacerations here, especially from jagged edges of glass (windshield and elsewhere), are obvious. Similarly, though rarely, contusive lesions in front of the neck may cause dangerous injuries of the esophagus and the trachea. At this writing, the author has under observation an elderly man who, while sitting in the front right seat, was thrown forward against the windshield and dash during a forward type of carom collision and received both a "whiplash" of the neck and a mild but notable contusion of the laryngeal prominence, with some frightening reactions but no real respiratory difficulties.

The classic mechanism of whiplash is a backward thrust of the head and the neck during a rear-end collision (with the patient sitting in the forward car) before the inertia of the head has been overcome, which is followed by an oscillating forward snap of the head and the neck before coming to rest. The same thing happens in reverse from a forward type of vehicular impact, and variations happen from side or even carom forms of impacts. The body reactions to sudden changes in velocity are dependent upon a number of factors. An occupant in a heavier vehicle, which is under way at the time of impact from behind by a lighter one, will be thrust forward. The jolts in both cars will vary also if one vehicle is at rest. Then, occupants may be thrown forward violently on to the steering controls and other forward structures.

The degree of protection afforded by the elastic limits of the soft tissues seems small, considering the magnitude of the forces acting on these structures in these accidents. Ordinarly, the limit of human tolerance for these stronger shocklike sprain-producing impacts is considered to be reached when disruption of tissue occurs. Do irreversible tissue changes occur when elastic, not structural, deformation of the tissue is exceeded? Probably not when the resistance of the bones and the joints remains above the breaking point. On the other hand, under excessive stresses, when these protective bony checks give way, actual rupture or yield of the soft parts may occur. Between these two extremes—sprains versus fracture—must reside other gradations of strain effects, even including subluxations and dislocations.

In the present series of 23 cervical sprains, 20 were adults, the majority being women: 11 were drivers; 6 were front, 3 back seat and 3 unclassified occupants. Seven resulted from rear-end collisions; 4 resulted from forward impacts; 2, from roll-overs; and 1, from forcible ejection. The impacts were recorded in only 34 accidents of the entire series of cervical injuries. There were 10 forward collisions with 7 soft tissue and 3 skeletal lesions; 9 rear-end impacts with 6 soft tissue and 3 skeletal lesions; 9 roll-overs with 5 soft tissue and 4 skeletal injuries; 3 driver dislocations with skeletal lesions only; 1 train collision with soft tissue injury; and 2 forcible ejections with 1 each of soft tissue and skeletal injury.

In the series as a whole, dislocations caused head impacts with the top of the vehicle. Injuries occurred at that time for most of these people. However, in some persons the injuries were incurred during rebound movements of the body and secondary impacts with interior structures. In other words, both positive and negative g forces operated in these vertical body displacements.

Practically all of the subluxations noted in this series were rotary and minimal in extent; and affected the atlanto-axial junction in 9 out of the 10 cases listed. Is this irrefutable evidence of trauma? The torsional qualities of these lesions place them below, or at best at the level of, sprains from the standpoint of degree of morbidity. The 10th subluxation in this series affected the 6th cervical segment. The detection of subluxations some-

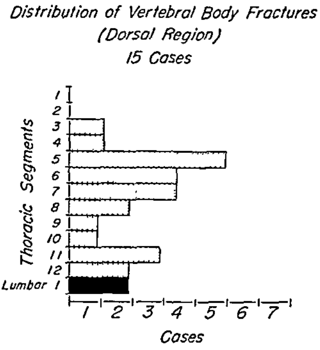


FIGURE 38

times requires laminographic investigations. In this series actual fractures affected the vertebral bodies in 50 per cent of cases, and *the secondary processes in the other 50 per* cent. The spinal cord was not involved in any case. It is more difficult to evaluate injuries of the secondary processes. In this series, involvement was as follows: lateral process at C-4, spinous process at C-6 (so-called shoveler's fracture), right pedicle at C-6 and transverse process at C-7. Injuries of the secondary processes may be confused with congenital or older lesions (nonunions), and this was the case in one or two instances in this series. Laminography may be helpful in this respect. The levels of fracture involvement in this small series was: 1st cervical, 1 case; 2nd cervical, 2 cases; 4th cervical, 2 cases; 5th cervical, 1 case; 6th cervical, 2 cases; and 7th cervical, 2 cases.

Very few disk lesions were suspected in these acute injuries of the cervical region.

INJURIES OF THE DORSAL SPINE

Of the 35 cases with dorsal injuries, 11 were not complicated by injuries elsewhere in the body (31%) Practically all the soft tissue injuries were contusions. The absence of lacerations and sprains is to be noted. It should be remembered that the dorsal spine may be exposed to considerable torsional and side-bending stresses in spins, skids and

TABLE 26. EXPERIMENTAL EVALUATION OF PORTION OF THE TOTAL BODY WEIGHT SUPPORTED BY THE INDIVIDUAL VERTEBRAE*

Vertebra	Values in Percentage of Body Weight
Dorsal 5	21
6	25
7	29
8	33
9	37
10	40
11	44
12	47
Lumbar 1	50
2	53
3	56
4	58
5	60

* Ruff: German Aviation Medicine, World War II, Department of the Air Force, U.S Government Printing Office, Washington, 1951.

TABLE 27. BREAKING LOAD (IN KILOGRAMS) OF VARIOUS VERTEBRAE*

Vertebra	Kg.
Dorsal 8	593
9	676
10	740
11	771
12	797
Lumbar 1	812
2	873
3	980
4	983
5	1040

* Ruff: German Aviation Medicine, World War II (The present table modified, in that it shows averages for age groups between 19 and 46 years of age, as shown in Ruff's original table)

roll-overs. Chronic dorsal backache is uncommon. In this series there were 16 contusions and 4 bruises and abrasions. Chief interest centers in the 15 cases with fractures of the vertebral bodies.

The violences to which the body is subjected in the various impacts would result undoubtedly in more fractures of this area were it not for the buffer action of the thoracic cage. However, the frequency with which patients who have impacted the steering controls (or any other forward structure) with their chests—with and without resultant fractures of the ribs and/or the sternum—and complain of severe dorsal pain, attests to the magnitude of the forces that are transmitted to the spine. The complaint of dorsal pain is sometimes the chief complaint in patients at the time of admission to hospital with rib fractures, but it clears up shortly unless there are demonstrable fractures of the dorsal or the lumbar spine.

Interestingly enough, in this series of 15 fractures of the dorsal spine, only 2 persons had both fractures of the dorsal spine and the ribs concurrently. In a 55-year-old woman, there was fracture of the 5th and the 6th dorsal vertebrae, together with fracture of the 4th rib and the sternum In the second, a 25-year-old drunk driver, there was fracture of the 12th dorsal vertebra, together with chest and extremity fractures. The evidence that the chest absorbs considerable amounts of energy to spare the dorsal spine seems to be strong.

In the present series of dorsal fractures, 26 vertebral bodies were affected in 15 persons. Multiple vertebral fractures result from the mild arcuate curve of the dorsal spine, which transmits forward flexion forces to more than one segment This is attested to by the fact that the most frequent areas of fracture were the 5th, the 6th and the 7th segments (Fig. 38), which required the proper kind of mechanical setup—best attained from the sitting posture. Second in frequency were the 11th, the 12th and the 1st lumbar segments, which were more in keeping with the traditional frequencies of vertebral fractures noted in the literature. Several other statistical factors were of interest.

Two persons were aged from 1 to 20 years; 8 ranged from 20 to 45; 4 from 45 to 65 years; only 1 was over 65 years of age. Twelve of these people were in the front seat (7 drivers); 2 sat in the back seat; and 1 was unclassified. The principal impacts were stated in 7 cases: roll-over, 1; ran off the road, 1; forward vehicular collisions, 3; and forcible ejection, 2.

BIOMECHANICS

Ruff reports on Geertz's experimental work regarding the breaking of the individual vertebrae, energy absorbing of the spinal column and percentage of the superincumbent weight supported by the individual vertebrae (Table 26). Analyses revealed experimentally that L-1 has the lowest tolerance for acceleration; it is between 23 g and 18 g. Ejection seat tests for pilots established the spinal tolerances of the neck and the lumbar areas to be 20 g for a duration of 0.1 sec. Tests that developed about 26 g for 0.005 sec. with 4 subjects resulted in spinal fractures in 3 of them, thus establishing this order of g force as the probable extreme upper limit of tolerance on the basis of the single individual who did not receive an injury (Table 27). Petter recorded that it required about 30 pounds of compressive force to return intervertebral disks to their original vertical diameter after they had expanded spontaneously from release of superincumbent weight, and even more so after section of the annulus. Moffat advanced the interesting theory that the disks hypertrophied in response to osteoporotic weakening of the vertebrae, especially after pathologic fracture of these vertebral bodies.

The advent of cervical and lumbar sprains focuses attention upon the stresses to which soft skeletal tissues are exposed, i.e., the tensile strength or the limits of elasticity of these structures. Pioneer studies on transplanted fascia lata were made in 1924 by Gallie and Lemesurier, who found that this

tissue retained its viability after 2 years. Gratz (1931) reported further studies on this tissue by engineering methods of testing the tensile strength. He found that the ultimate tensile strength of fascia lata was approximately 7,000 pounds per sq. in., or nearly as *strong as soft steel, weight for weight.* McMaster's clinical and experimental observations on tendon injuries and tensile strength also confirmed the great relative strength of this structure which yields only at bony attachments or musculotendinous junctions under normal conditions of stress. In the face of such demonstrable strengths of isolated soft tissues, why are sprains of the cervical and the lumbar regions so prevalent?

PROPHYLAXIS

Regarding higher tolerances to linear decelerative forces in rearward facing seats, forces transmitted via the back rest of the seat literally are distributed over a much larger area. Ruff found that for the abdominal and the shoulder straps, the shock absorption area is 300 cm.^{2} (48 sq. in.); for the backward arrangement it is 1300 cm.^{2} (208 sq. in.). Interestingly enough, he abandoned his experiments at orders of 28 to 30 g's tolerances for approximately 0.1 sec. when engineers said that it was impossible to increase the resistance of the individual seat and seat attachments in the then existing aircraft without involving almost insuperable difficulties in design (resistances of 2,000 to 2,500 Kg). Thus, then, even as now, does structural, not human, limits determine the standards of structural factors of safety and design.

The normal gravitational pull or head to foot (inertial reaction) is called positive g, while negative g is foot to head. The following values are said to represent the approximate limits of the body to the various g forces: positive g is 20 g for 0.1 sec.; negative g is 10 g for 0.1 sec.; transverse g (chest to back) is 55 g for 0.01 sec.; and, transverse g (back to chest) is 60 g for 0.01 sec.

Of course, these values apply to inertial responses to centrifugal forces experimentally produced or experienced in flight.

BIBLIOGRAPHY

Davis, Arthur: Injuries to the spine, *in* Bancroft, F., and Marble, H.: Surgical Treatment of the Motor-Skeletal System, ed 2, Philadelphia, Lippincott, 1951.

Gallie, W. E., and LeMesurier, A. B.: The transplantation of fibrous tissue in the repair of anatomical defects, Brit. J. Surg 12:289, 1924-25.

Gratz, C. M.: Tensile strength and elasticity tests on human fascia lata, J. Bone & Joint Surg. 13:2, 1931.

McMaster, P. E.: Tendon and muscle rupture, J. Bone & Joint Surg. 15:3, 1933.

Moffat, Barclay W.: Enlargement of the intervertebral disc associated with decalcification of the vertebral body: a compensatory hypertrophy, J. Bone & Joint Surg. 15:679, 1933.

Petter, Charles K.: Methods of measuring the pressure of the intervertebral disc, J. Bone & Joint Surg 15:365, 1933.

Ruff, Siegfried. Acceleration, less than one second, *in* German Aviation Medicine, World War II, vol. 1, p. 584, Washington, D. C., Department of the Air Force, 1950.

Schneider, Justus Protective measures, especially spinal fractures, *in* German Aviation Medicine, World War II, Washington, D. C., Department of the Air Force, 1950.

12

Acute Injuries of the Lumbar Spine and the Pelvis

INJURIES OF THE LUMBAR SPINE

Of the 61 persons with injuries of the lumbar area, 9 (or 15%) were uncomplicated by other injuries. Soft tissue injuries were twice as frequent as skeletal ones. Of the former, 17 were contusions, 2 abrasions (no lacerations), 19 sprains and 1 disk herniation. This last occurred in an elderly female who received, in addition to the low back pain, a whiplash subluxation of the cervical spine. Both levels were complicated by osteoarthritis of the spine. Operation did reveal a herniated lumbosacral disk, which was removed. The result was good. In this connection, a young man who had been operated upon for herniated lumbosacral disk (interlaminar technic, without fusion) was involved in an automobile accident in which his low back was wrenched about 2 years later. He recovered from his sprain without further disability in regard to his old disk lesion.

Of the 19 lumbar sprains, 12 were in women and 7 in men, and they were in the following age groups: under 20 years, 2; from 20 to 45, 11; from 45 to 65, 6. Ten were drivers, the rest, passengers—2 in the front seat, 3 in the back seat, and 4 unclassified. The principal impacts included: 7 forward vehicular collisions and 1 each of forcible ejection, struck from side, rear end, roll-over, off the road and collision with train.

In the 24 fractures of the lumbar spine the sexes were fairly evenly divided. Their ages ranged as follows: 1 to 20, 2 cases; 20 to 45, 15 cases; 45 to 65, 4 cases; and over 65, 3 cases. Distribution according to seating was: 12 drivers; and 12 passengers—5 in the front seat, 3 in the back seat and 4 unclassified. The principal impacts included: forward collisions, 9; forcible ejection, 3; roll-over and off the road, 2 each; struck from the side, 1; and driver dislocated, 1.

The frequency rates of sprain, fractures of the secondary processes (through indirect action of muscles) and vertebral body fractures again brings to mind a progressive series of events from the standpoint of increasing severity of forces and their effects. These various injuries were about evenly divided and produced by more or less *similar* vehicular impacts. There were more of the forward types of impacts, however, in relation to the fracture group.

From the standpoint of mechanism of injury which usually is attributed to fractures involving the vertebral bodies and the secondary processes, one is impressed immediately by the relative localization of the former as compared with the widespread types of injuries that affected the secondary processes. In other words, the forces operative in the production of vertebral fractures acted about a more or less localized fulcrum. On the other hand, fractures of the secondary processes would indicate indirect forces exerted through a considerable range of musculature and tendinous structures in an indirect manner. Consequently, fractures of the secondary processes of the lumbar vertebrae (especially the transverse ones)

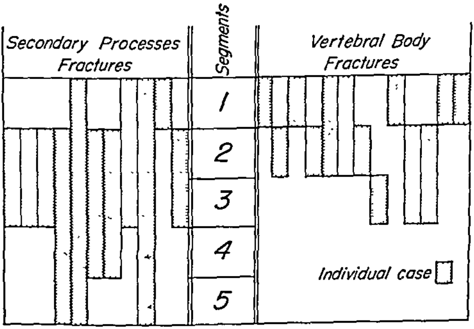
cannot be the innocuous injuries some authors state them to be. These fractures would seem to bridge sprains to fractures of the vertebral bodies (Fig. 39). There were no injuries to the spinal cord.

INJURIES OF THE PELVIS

Of the 55 persons with injuries to the pelvic regions, 12 (or 22%) were not complicated by other injuries. Eight of these received fractures; 2 had soft tissue lesions and 2 had joint lesions. The pelvic region was unique in that the skeletal injuries outnumbered the soft tissue ones. Thirty-nine received fractures. Among the soft tissue injuries there were 10 contusions and 4 abrasions, which affected chiefly the buttocks and the hip regions. However, 3 persons received lacerations of the buttocks, the perineum and the genitalia. The last 2 areas usually are associated with very severe motorist accidents. Here, both were incurred by women who were sitting in the right front seat and were ejected forcibly on impact. The first, a 20 year old, was thrown into a wheat field and received perineal lacerations, as well as lacerations of the face and the extremities. The second was 38 years old; she was thrown on to a stony road and received severe lacerations of the labia, which were grossly contaminated by dirt and other foreign bodies. Laceration of the buttocks occurred in a child involved in a collision between a train and an automobile.

With regard to skeletal injuries of the pelvis, 39 persons received 44 fractures of the pelvic region (including trochanteric). Injuries may be classified into 3 categories: those involving the innominate bones; those in the hip regions; and those in the junctions in front and behind (including the coccyx).

There were only 4 posterior injuries: 2 affected the sacro-iliac joints; 1, the sacrum; and 1, the coccyx. Not all sacro-iliac lesions were associated with displacements at the symphysis pubis. The paucity of sacro-iliac lesions is to be remarked. The sacrum was fractured, the coccyx was subluxated, and the sacro-iliac joints were sprained in one



DISTRIBUTION OF LUMBAR FRACTURES
(In present series)

FIGURE 39

and subluxated in the other. Nevertheless, posterior injuries should be evaluated carefully, whether or not they are complicated by urinary difficulties, since the presacral plexus of nerves may be injured. Sacral fractures may be incurred from forcible ejection. Likewise, severe fractures of the pelvic ring, with *sacro-iliac displacement*, also require large magnitudes of force to produce them.

In the *fractures of the pelvic ring or the innominate bones*, a single bone was affected in 12 persons; 2 bones, in 2 and 3 bones, in 3. There were no fractures due to indirect or muscular causes (anterior superior iliac spines or ischial epiphyses). This contrasts sharply with what happened in the lumbar region, and may be explained by the fact that only 4 of these patients were under 20 years of age, and only one of these was young enough to have had an epiphyseal avulsion. Moreover, the relaxed sitting position would militate against avulsions at the anterior part of the pelvis. Women predominated in regard to pelvic fractures. There were 3 drivers, 8 guest passengers (chiefly in the front seat), and the rest were unclassified. The principal impacts were recorded in only 7 instances: 4 forward collisions, 1 ejection, 1 train-automobile collision and 1 off-the-road accident.

It is to be remarked that in the consecutive series of motorist survivors on which this series of pelvic injuries is based, there were no internal pelvic injuries. All severe fractures of the pelvis, especially those involving the anterior arch (pubic bones), demand a most careful evaluation of the genito-urinary situation and prompt careful treatment relative to injury or misfunction. Look for urinary bladder injuries in people who have been ejected and exhibit injuries of the forward pelvis.

The large number of injuries related to the hip joint are to be remarked in Table 28. The 3 trochanteric fractures affected 3 male drivers—1 in his early twenties, the other 2 in their early forties. Two received multiple fractures; the youngest was not thus

TABLE 28. INCIDENCE OF FRACTURES AND DISLOCATIONS OF THE PELVIS

BONE OR JOINT AFFLICTED	NO.
Fracture dislocation hip	10
Fracture pubis	9
Fracture ischium	6
Fracture ilium*	5
Fracture acetabulum	5
Fracture trochanter femur	3
Simple dislocation hip	2
Fracture sacrum	1
Subluxation sacro-iliac joint	2
Subluxation coccyx	1
	44

* 1 compounded

complicated. In 1 case, there was no displacement, and healing took place uneventfully. The other 2 required open reduction and internal fixation. The fracture in the youngest patient (fixed internally) was extremely slow in healing. There were no transcervical fractures of the femur in this series, although such have been treated by the author—1 a young woman of 20.

There were 18 persons with injuries involving the hip joint proper: 10 were fracture dislocations; 5 affected the acetabulum without dislocation (outwardly); and 3 were simple dislocations without fracture. Thus associated, it becomes obvious that these injuries also represent progressive degrees of force applied in approximately the same manner. Urist considered these lesions as controls, one against another.

The 3 simple dislocations occurred in 2 men aged 28 and 39 years, both drivers. The third was in a woman aged 56 years, a front seat guest passenger. Only 1 injury was uncomplicated by others; the 2 remaining ones incurred multiple other injuries. One accident involved a head-on collision, 1 was an off-the-road occurrence, and the other was unknown.

The 5 acetabular injuries all involved men whose ages ranged between 29 and 70 years,

4 of whom were under 40 years of age. Three were drivers, 1 was a passenger in the front seat, and 1 was unclassified. Three were forward collisions; the other 2 were unknown. Two were chip fractures of the upper rim of the acetabulum, 1 a fissure fracture of the floor of the acetabulum and actually were so-called central dislocations of the femoral heads. All 5 persons incurred rather severe multiple injuries elsewhere in the body.

There were 10 fracture dislocations of the hip joint—in 6 men and 4 women. All of the dislocations were posterior ones. Only 3 were uncomplicated by other injuries, the rest incurred rather severe multiple injuries elsewhere. Eight were drivers, and 2 were front seat guest passengers The principal impacts were recorded in 6 instances: 5 forward collisions (3 head-on vehicular, 2 with fixed objects); and 1 collision with a train.

In the series of hip injuries as a whole, there were 8 on the right side and 12 on the left side.

There was 1 case of fracture dislocation of the hip with sciatic paralysis. It occurred in an adult woman who had been sitting in the right front seat during a forward crash in which her driver husband had received serious head injuries. An open reduction was done, after which aseptic necrosis of the femoral head was noted. However, both complications cleared up spontaneously in about 2 years

BIOMECHANICS

Both Steindler and Evans review the older and more recent literature with regard to force conditions involving elastic limits and fractures of the pelvic bones. Evans and Lissner (1955) expanded the Wayne University school of study regarding stress coat technics showing tensile deformations to the pelvis and the lower spinal column. As little as 33 in. pounds of energy input in the ischial tuberosities caused tensile deformations such [illegible] occur in any elastic body. The [illegible] over the buttocks proved to be an excellent energy-absorbing material. The various static and dynamic loadings causing the various fractures of the pelvis indicated that these injuries were due to bony failures from tensile stresses. Moreover, the various types of pelvic fractures produced experimentally were similar to those reported in the clinical literature.

PATHOMECHANICS

The strength of the spinal column is said to depend upon the breaking load of the vertebrae under pressure; the compressive strength, which is made up of the compressibility of the disk, deformability of the vertebrae and the elasticity of the upper part of the body; the portion of the body weight carried by the most heavily loaded vertebrae; and the time during which the acceleration is effective.

Very recently the author treated a rather severe compression of the 1st lumbar vertebra in a young adult male who had been sitting in the back seat between 2 other men during a forward impact of the car against an embankment at moderate speed The driver escaped injury, as did the 2 other men in the back seat The elderly mother of the driver in the front received fractures of both upper ends of the humeri. The puzzle of the lumbar fracture in the man between 2 others who were unhurt was dispelled when it was learned that this man had had an arthrodesed hip for years. Thus, on impact, his lumbar spine was subjected to excessive stresses. This reciprocal relationship is well known to orthopaedic surgeons, who take it into consideration when making indications regarding arthrodesis of the hip joint. Fusion of the hip is to be avoided in those with arthritic lumbar spines.

By analogy, it is evident that the pivoting [illegible] at the hips afforded by properly fitting [illegible]lts reduces the total energy of head [illegible]gh centripetal dissipation of en[illegible] as some loss of it in the com[illegible] of the spine. Th[illegible] case also

illustrates the importance of good general condition of the individual at the time of crash and how one abnormal section of the body exposes another portion to excessive stresses, the chain being as strong as its weakest link.

PROPHYLAXIS

In regard to fractures of the vertebral bodies, compressive forces were responsible in all cases. Patients fell or slumped to the floor, with seat backs or other occupants on top of them, thus jamming and jackknifing the spine. One young patient had his father and mother fall on him with a crushing weight. "Packaging" certainly does not mean crowding.

Urist and others have demonstrated the close relationship between automotive design and certain injuries of the hip joint. In Urist's series of 58 such lesions, 40 of which occurred in jeep accidents, 27 were fracture dislocations, 16 were fractures of the acetabulum without dislocation, and 15 were simple dislocations without fracture. On the basis of experimental and clinical evidence, he reasoned that the peculiar design of the jeep and the *variable preimpact positions* assumed by its occupants definitely predisposed these people to the 3 types of hip injuries. Urist concluded that the blow might be received by the greater trochanter, or it might be transmitted through the femur from the flexed knee on the dashboard or from the foot on the floor board, or it might come from behind and be transmitted to the hip from the sacrum.

SUMMARY AND CONCLUSIONS

About 28 per cent of motorist casualty survivors in this *series received injuries of* neck, back and pelvis. Due to the basic sitting posture of automobile occupants, these parts of the body form an integrated whole, both from the clinical and the crash-impact points of view. Of special interest are the soft tissue injuries, mainly so-called sprains in the neck and the lumbar regions. For the most part, these are due to indirect inertial forces, in contradistinction to injuries from direct impacts. However, there is a close relationship between soft tissue skeletal injuries in these areas of the body, which are less obvious in other parts. From the standpoint of increasing severity of injury, sprains, subluxations, dislocations and fractures represent such a progressive series of events in the spinal situation. After fracture would come actual rupture of soft parts. Disk lesions bridge the gap between soft tissue and bony injuries from a mechanical viewpoint. Due to the close relationship that exists between these various structures, diagnostic and therapeutic questions are to be expected, and they must be resolved on the merits of the individual case.

From the orthopaedic standpoint, injuries of the hip joint probably disclose most convincingly the predisposition to injury that certain features of automotive design impose upon occupants.

Prophylaxis demands keeping the occupants in their seats by safety gear, in conjunction with adequate seat, seat back and gear *installations.*

BIBLIOGRAPHY

Bigelow, J. L.: The Mechanism of Dislocations and Fracture of the Hip, Boston, Little, 1900.

Carey, E J.: Studies in the dynamics of histogenesis, Radiology 13:127-168, 1929

Davis, Arthur G.: Injuries to the Spine, pp. 749-797, Philadelphia, Lippincott, 1945.

Evans, G. F., and Lissner, H. R : Studies on pelvic deformations and fractures, Anat. Rec. 121:141, 1955.

Ruff, Siegfried: Acceleration: less than one second, *in* German Aviation Medicine, World War II, vol. 1, pp. 584, Washington, Department of the Air Force, U.S. Government Printing Office, 1950

Sonnenschein, H. D.: Traumatic dislocation of the hip, J. Bone & Joint Surg. 11:96-99, 1929.

[illegible] of the Human [illegible] 1955.

[illegible] joint, Am. J. Surg. 74:586, 1947; J. Bone & Joint Surg. 30-A:699, 1948; Ann. Surg. 127:1150, 1948.

13

Injuries of Young and Old

The series of 661 motorist casualty survivors fell into 4 major age subgroups: those up to 15 years; from 15 to 20; from 20 to 60; and from 60 to 85 years of age. Figure 40 establishes graphically the relative incidence of motorist casualties from this standpoint. The adults whose span of years was 40 made up 67 per cent of the total number of patients. The remaining 33 per cent covered a 45-year span. A further breakdown established that 7 per cent of the injuries occurred in children; 16 per cent in teens; and 10 per cent in the aged. These percentages included both sexes (Table 29).

In this chapter will be considered the injuries received by 220 persons (33% of total series) who comprised infants, children, teens and those 60 years of age and over.

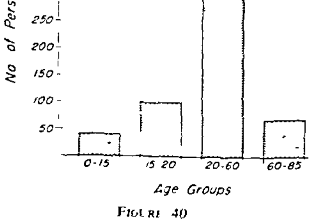


FIGURE 40

Table 30 lists the distribution of young and old motorist casualties according to seating. On the whole, there were 27.72 per cent drivers; 52.27 per cent guest passengers; and, 20.01 per cent unclassified occupants. The frequency rates of drivers in the various age groups was none for the children, 9.83 per cent for those from 15 to 16 years of age, 47.54 per cent for the 16- to 20-year-old group, and 42.63 per cent for the aged. In other words, the majority of these people had been dependent upon others for their safety, as was most notable in the 15- to 16-year-old group.

Figure 41 shows graphically the incidence (percentages) of injuries to the various body areas in children, teens and the aged. This

TABLE 29. DISTRIBUTION OF YOUNG AND AGED MOTORIST CASUALTIES ACCORDING TO AGES IN YEARS AND SEX

Age in Years	*Males*	*Females*	Total No.	%
Up to 2	1	4	5	2.25
2 to 5	2	1	3	1.36
5 to 10	9	7	16	7.27
10 to 15	10	11	21	9.54
15 to 16	14	8	22	10.20
16 to 20	49	33	82	37.27
60 to 65	14	16	30	13.54
65 to 70	3	12	15	6.77
70 to 75	5	7	12	5.45
75 to 80	4	6	10	4.54
80 to 85	2	2	4	1.81
	113	107	220	100.00

TABLE 30. DISTRIBUTION OF YOUNG AND OLD MOTORIST CASUALTIES ACCORDING TO SEATING

AGE GROUP	NO. OF DRIVERS	NO. OF GUEST PASSENGERS	NO. OF UNCLASSIFIED OCCUPANTS
60 to 85	26	27	18
16 to 20	29	31	22
15 to 16	6	12	4
up to 15	0	45	0
	61	115	44

curve slopes downward with a slight bulge at the facial and the chest levels. The sharp peaks at the face frequencies for the younger age groups is to be noted, particularly for children. Viewed from another angle, the peripheral parts of the body were injured in 85 per cent, 81.76 per cent and 65.53 per cent in children, teens and the aged, respectively. The body masses were more vulnerable in the aged, chiefly in the chest areas. This probably was due to loss of elasticity of these structures.

The paucity of internal injuries in the aged is to be remarked. There was 1 each of hemothorax and pneumothorax, 1 suspected cardiac contusion, 1 intracranial hemorrhage, 6 cases of shock, 6 cerebral concussions and 1 case involving both shock and concussion. Among the accessory findings were an active peptic ulcer, 1 chronic cardiac disease and 1 case of pulmonary atelectasis. The cerebral concussions occurred in 5 drivers and 1 rear seat passenger. Shock occurred in 5 front seat passengers and 1 driver.

In the children, there were no serious internal injuries. In the teen group there were 3 instances of brain injury, and a ruptured kidney in another one. Shock was prominent in 2 (about 4%) of the children and in 4 (about 4%) of the teens.

Table 31 establishes the nature of the injuries received and their frequency in the different body areas in children, teens and the aged. On the whole, there were about twice as many topical or soft tissue injuries as skeletal ones in every age class. The skeletal lesions were practically all fractures. There were relatively few sprains, subluxations and dislocations. In those under 2 years of age there was only 1 fracture—that of the skull. For the rest, there were only soft tissue injuries for this youngest category. From 2 to 5 years of age, the 3 patients received a fracture of the femoral

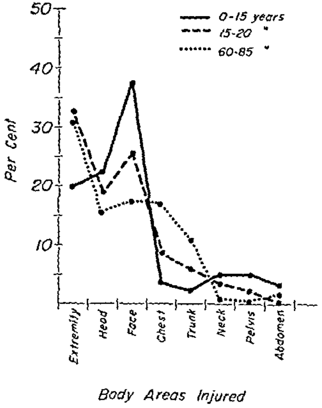


FIGURE 41

TABLE 31. NATURE OF INJURIES RECEIVED BY CHILDREN, TEENS AND THE AGED

PART OF BODY	CHILDREN Topical No.	CHILDREN Skeletal No.	TEENS Topical	TEENS Skeletal	AGED Topical	AGED Skeletal
Extremity	6	10	35	3	29	35
Head	14	4	37	2	25	3
Face	21	9	34	18	26	5
Chest	2	1	12	6	12	18
Trunk	1	1	7	6	4	4
Neck	3	1	1	8	1	1
Pelvis	3	1	1	4	0	1
Abdomen	3	0	1	0	3	0
Multiple	6	0	10	0	18	0
	59	27	138	47	118	67

shaft, a nasal fracture and body contusions. The fifteen patients between 5 and 10 years got 12 fractures. Twenty-one of those from 10 to 15 years had 14 fractures. It should be noted that approximately 50 per cent of the lesions were skeletal in the young but a little more than that in the aged.

Fractures in the teens paralleled more or less those of the series of 661 motorist casualty survivors in general.

On the other hand, in the aged, some differences were notable. Of special significance was the preponderance of skeletal injuries over soft tissue ones in regard to the extremities and the chest areas. The 6 skeletal lesions, other than fractures, included 1 sprain each of ankle, wrist, neck and lumbosacral junction and 1 dislocation each of the shoulder and the acromioclavicular joints. Fractures of skull, face, chest, spine and pelvis were distributed among 25 persons—16 women and 9 men. Two of the skull fractures involved the frontal bones and 1 the base The mandible was broken twice and the nose 3 times; teeth were loosened once. The pelvic injury resulted only in a chip off the acetabulum All spinal fractures involved the lumbar region—the vertebral body in 1 case and the secondary processes in 2 cases.

Among the fractures of the ribs, single ones were affected in 3 instances, none involving the 1st rib. Multiple rib fractures occurred on 1 side in 16 cases and bilaterally in 1 case. The absence of sternal fractures in aged survivors is to be noted. Fractures of the ribs were received by 5 drivers and 9 right front seat passengers (again emphasizing vulnerability versus seating).

In children, fractures of the arms and the legs were about evenly divided. This was due to lack of leg bracing and tumbling kinematics of small children. But in the aged there were twice as many fractures of the lower extremities as of the upper ones Bracing probably accounts for the preponderance of lower extremity lesions. For the latter, there were 7 fractures in the shoulder, 3 in the elbow, 2 in the forearm and 1 in the wrist. For the lower limbs, there were 8 fractures in the knee, 7 in the ankle, 4 in the leg, 2 in the foot and 1 in the thigh. The absence of fractures and fracture dislocations at the hip is to be remarked. There were 12 men and 15 women in this fracture category of the aged. Among the drivers here there were 9 men and 1 woman.

The question was raised naturally as to whether or not older persons were more vulnerable to fractures under crash conditions than younger ones (see footnote, p. 323).

The incidence rate of fractures for children, teens and the aged was 45, 55 and 61 per cent, respectively. However, from the standpoint of total number of fractures incurred in each class, the rates were 18, 42 and 40 per cent, respectively. In other words, older people with more brittle bones are more liable to fracture. But their rate of fracture based on multiplicity of fractures was a bit lower than that for teens but vastly greater than that for children. More specifically, older people are more apt to get fractures of the extremities and the ribs, but they are less likely to receive fractures of the facial, the pelvic and the spinal bones during vehicular collisions.

Another criterion in regard to tolerances of older people to crash forces was noted. From the rate of survival and the relatively low frequency of fractures from some rather severe types of collisions, the inference is obvious that healthy older persons manifest remarkable capabilities for absorbing impact energies as well as inertial forces that are generated under crash and/or upset conditions of ground vehicles. Good physical condition, rather than chronologic age, must be a most important factor in determining impact tolerances.

DISCUSSION

One hundred and twenty-five, or 19 per cent, of this series ranged in age from infancy through the teens. Actually, 74 per cent were teens. The largest section of the teens were 17 years of age (30%). Seventeen per cent were between 13 and 15 years of age. Seventeen per cent were aged 16—the legal age for driver licensure in the state of Missouri. One 15-year-old girl and 5 16-year-old boys were drivers (impacteens).

PRINCIPAL IMPACTS

The type of accidents in which children and teens were injured were recorded in 44 instances. They included: forcible ejection from the crash vehicles, 14; head-on collisions, 9; roll-overs, 7; and the remainder involved collisions with fixed objects, running off the road, rear end, train-vehicle; and simple dislocation of the driver by a bump in the road (in 1 instance). In other words, these represented the run-of-the-mill, or garden, variety of accidents suffered by all other segments of the motoring community. Moreover, no mechanical failures were noted as causes of the accidents. However, outstanding was the high percentage of children, especially the little ones, who were ejected forcibly from the crash vehicles.

PEDIATRIC PATHOLOGY

Case 1. Female, aged 3 years, died 7 hours after admission to the hospital. She was in the back seat with several brothers and sisters when her car crashed into the back end of a stalled truck. All told, there were 9 persons in her car, and all were hurt in varying degrees. She was the only fatality, and died in hyperpyrexia. Autopsy revealed only focal areas of cerebral petechial hemorrhages—without skull fracture. There was already some evidence of passive congestion of the lungs.

Case 2. Female, aged 11 years, died 8 hours after admission to the hospital. She was ejected from the rear of a pickup truck and struck her head on the pavement. Autopsy showed separation of the cranial suture lines at the frontal and the temporal areas. There were multiple focal hemorrhages in the mid-brain, subarachnoid hemorrhage in the brain stem, and moderate edema and blood in the ventricles. In addition, she had received a severe contusion of the heart. The left optic nerve had been severed. There were focal hemorrhages in the spleen and an advanced bronchopneumonia.

Case 3. Female, aged 3 years, died 3 hours after admission to hospital. She had been ejected and run over. The right leg had been practically amputated; the left tibia and fibula also fractured The spleen was ruptured, the right lung lacerated and complicated by hemothorax. The brain was contused but without skull fracture, and showed subarachnoid hemorrhages and edema with compression. She died in hyperpyrexia. Among the complications worthy of special mention were a lower nephron nephrosis, secondary shock (marked congestion of all the viscera), fatty liver, fat embolism of the lungs and a marked tracheo-

bronchitis, with considerable exudate in the bronchi.

Here, then, concentrated into 3 small bodies are all the most deadly of the killers of motorist casualties—multiplicity of peripheral injuries, internal injuries and complications

By contrast, fatalities in the aged focus chief attention upon thoracic and intrathoracic injuries, of which those of the mediastinum already have been quite fully discussed in the chapter on pathology.

AIDS TO RECOVERY AFTER INJURY

Children and aged motorist casualties, being at opposite extremes of the human life span, differ very little from each other basically regarding both their reactions to injury. In some respects, however, the limits of factors of safety are different. For example, water and electrolyte balance may be even more difficult to maintain in children than in the aged, who traditionally are dehydrated to begin with. Azotemic and uremic states are a more constant threat in both of these age extremes than is to be expected ordinarily, except in the frank crushing injuries of the extremities in any age subgroup. Azotemia (extrarenal) is characterized by an abnormally high level of urea or other nitrogenous waste products in the blood.

Uremia (renal) shows the presence of urinary constituents in the blood and the toxic condition produced thereby—muscular weakness, dyspnea, mental disturbances, nausea and vomiting, muscular twitchings, stupor and coma. Acidosis is manifested by fast respiration, coma, strongly acid urine with excess ammonia—becoming scanty—anuria; usually dehydration; and lowering of the CO_2 combining power (normal 55-80). Signs of alkalosis include shallow respirations; lethargy, prostration, nervous irritability, progressively developing toxemia; dehydration; scanty urinary output, alkaline urine with a decreased ammonia; twitching of facial muscles, tetany; increased CO_2 combining power over 80 (tetany may be expected as this value approaches 100); fall in plasma chloride and rise in nonprotein nitrogen.

A most distinctive difference exists in the treatment of fractures of the extremities in the young and the old. In the former, open reductions, if any, always are performed by choice. In other words, conservative methods of treatment are to be preferred for children. On the other hand, open reductions and internal fixative means are methods of necessity in regard to fractures in the aged, because very frequently operative technics permit early mobilization of these old people. In these situations, movement literally is life, since decubiti and pulmonary hypostasis are avoided thereby. In other words, operative measures of reduction and fixation actually become conservative methods in regard to the older fracture patients.

IMPLICATIONS IN REGARD TO ACCIDENT PREVENTION

The social and the philosophic implications of injuries of the young and the old may be likened to a giant teeter-totter, the balance being threatened constantly by the young and the old at opposite ends of the board. Balance cannot be attained between youthful thoughtlessness on one hand and indifference of the aged on the other. It needs a stabilizing influence at the center, which can be achieved by the mass of adults who normally make up this sector of motorist casualties. This is not as easy as it may seem on first thought. Moreover, during moments of indecision the teeter-totter becomes a merry-go-round, at which times centrifugal influences are exerted one way or the other, either toward the left or the right — toward infantility or senility of thought, action or attitude.

Adults may take 2 courses: the first in regard to their own driver education and training, and the second in regard to their chil-

dren's education in motorist survival. At the same time, more interest is devoted to local, county, state and national laws or codes that deal with traffic problems. It will be found that achievement at the community level will spread rapidly outward from community to community.

At the other extreme, gerontols who have been driving often make up in experience what they may lack otherwise. This cannot be said for the oldster who gets behind the wheel for the first time in his dotage. It may be wise to place these people in categories that place some restrictions on their driving privileges, as is done in the case of students qualifying for their pilot's license. For example, these neophytes are not allowed to carry passengers until they have proved their ability and worthiness to take on this responsibility.

In regard to pilot licensure, a movement already is afoot to lower somewhat the physical standards for private flying. In other words, Coates has suggested a modification of the physical criteria that now are used for pilot licensure in regard to private flying only. These people are to be placed in a physically defective classification similar to that of the student pilot, who is not permitted to carry passengers. Thereby, persons with physical disabilities that are not debilitating could be licensed to fly but could not carry passengers. Ironically enough, Coates' idea is based on the fact that such people would have a better chance for their lives during air travel than they now have on the ground, which seems like stretching the point a bit.

CONCLUSIONS

The lessons of injuries to the proximal and the distal fringes of the motoring population—young and old—are more philosophic than scientific, more psychological than medical. These people, while they make up the warp and the weft of the tapestry of motorist injuries and safety, do not furnish the design. That is made up by the mass of adults in between the young and the old. To paraphrase Shakespeare, the fault is in ourselves. I do not mean to underestimate the faults of youth or the infirmities of old age as they relate to driver proficiency; I wish only to point out that it gets us nowhere to seek a scapegoat—young, old or in between. For too long the story of traffic safety has been made attractive with such sugar-coated placebos. The thing cuts across all citizens in any community.

In regard to the injuries, the chief differences between the young and the old are in the elastic limits of their tissues.*

*Boris Tourin (Cornell Automotive Crash Injury Research) wrote in a personal communication: "In an epidemiologic-type study of head injuries, based on 2,253 occupants of 1,000 automobiles, it was found that there was a significantly lower frequency of injuries to the head among persons aged up to 20 years as opposed to those who were over 20 years of age." He stated further that this finding was due to the fact that most of the younger occupants had sat in the back seat, which has been found to have a much lower injury potential than the front one.

BIBLIOGRAPHY

Coates, Thomas A.: A restricted medical certification for civilian pilots, J. Aviation Med. 26:70, 1955.

Kulowski, Jacob: Motorist injuries among persons over 60, Geriatrics 10:425, 1955.

———: Motorist injuries to children, J. Pediat. 47:696, 1955.

Moon, Virgil H.: Renal deficiency associated with secondary shock, South. M. J. 49:180, 1956.

Rice, C. O., *et al.*: Lower nephron nephrosis (2 papers), Am. J. Surg. 90.547, 558, 1955.

Snyder, H. C.: Intravenous fluid therapy in children, J A.M.A. 158:1004, 1955.

14

Residual Disabilities: General Skeletal

Data for this chapter were extracted from the medical records of 215 motorist casualty patients who were seen consecutively in the author's office from late in 1949 through July, 1955. All these people had chronic complaints of the motorskeletal system at periods of time ranging from several weeks to a year or longer after accident. All these persons were ambulatory. The vast majority had been hospitalized by other medical attendants during the acute phases of the alleged accidental injuries. The general aspects of these chronic or residual disabilities will be discussed in this chapter. It will deal chiefly with lesions of the extremities. In the following chapter those of neck and back or lumbar region will be discussed.

GENERAL STATISTICS

Table 32 establishes the age distribution of 215 motorist residual disabilities of the motorskeletal system. Eighty-one per cent of this series involved the working span of life (20-65 years), and 58 per cent of these were in the age group (20-45) when degenerative conditions are said to be minimal. Fifty-five per cent were males. It is to be noted that only 5 per cent of these people were aged; 14 per cent were young persons. These figures were a little higher in regard to acute injuries in the young and the old (10% & 16%, respectively). The universal effects of mechanical force in any guise and the age incidence noted in this series link these disabilities to those that occur most frequently in industry, since it has been established that even in automobile workers there are more off-the-job accidental injuries—especially those involving going to and returning from work—than on-the-job ones.

TABLE 32 AGE DISTRIBUTION AMONG 215 MOTORIST CASUALTY DISABILITIES OF THE MOTORSKELETAL SYSTEM

AGE RANGE	No	%
Up to 20 years of age	31	14
From 20 to 45	124	58
From 45 to 65	50	23
65 and over	10	5
	215	100

Table 33 lists the types of vehicles which

TABLE 33. TYPE OF VEHICLES INVOLVED AND THEIR FREQUENCY

COLLISIONS BETWEEN VEHICLES	No.	NONVEHICULAR CRASHES	No.
Passenger cars	83	Passenger cars	37
Car and truck	29	Trucks	11
Truck and truck	2		
Trailer and car	2		
Truck and train	1		
Car and train	2		
Bus and car	2		
Car and snowplow	1		
Hayrack and car	2		
Car and ambulance	2		
	126		48

TABLE 34. PRINCIPAL IMPACTS

	No.	%
Forward collision	50	30
Rear end collision	41	25
Roll-over	25	15
Right angle collision	24	15
Carom (oblique)	9	5
Ran off road	9	5
Collision with fixed object	8	5
	166	100

TABLE 35. PATHOLOGIC ANATOMY AMONG 215 MOTORIST MOTORSKELETAL LESIONS

BODILY REGION INVOLVED	No.	%
Cervical	66	28
Low back	65	27
Lower extremity	67	28
Upper extremity	26	11
Miscellaneous	13	6
	237	100

were involved in this series of accidents and their frequency rates. Approximately 70 per cent involved passenger cars, and 30 per cent were commercial carriers of one kind or anther. Seventy-five per cent involved 2 or more drivers.

Of the vehicular collisions, about 35 per cent involved commercial carriers. Furthermore, of the nonvehicular collision type of accident, about 25 per cent also involved commercial carriers. Viewed from another angle, however, 92 per cent of all the accidents in this series involved passenger cars in one way or another. This statistical digression has implications in regard to first aid, which has been stressed in an earlier chapter. Clearly, both commercial carriers and private transport have a rather large stake in motorist safety.

Table 34 establishes the principal impacts regarding 166 accidents. The large number of front end and rear end collision types of impacts is to be remarked. Nonvehicular types of impacts were relatively uncommon. It is evident that all types of principal impacts result in residual disabilities of the motorskeletal system. Eighteen persons were ejected forcibly during impacts. In 4 cases the doors opened in cornering; the remainder occurred with roll-overs, spins and rear end and other types of vehicular and nonvehicular collision impacts.

Eighty-eight per cent were in the front seat, of whom 53 per cent were drivers. The relative incidence of men and women approximated that for the entire series of 661 survivors. Keeping in mind certain peculiarities of insurance liability laws, rules and regulations, the medicolegal involvements in this series was distinctive. In this series of 215 persons, 40 per cent required some kind of disability rating. From the standpoint of seating, the frequency rates of those with physical disability ratings was as follows: drivers, 53 per cent; front right seat passengers, 31 per cent; back seat passengers, 16 per cent.

CLINICAL PATHOLOGY

Table 35 focuses attention upon the frequency incidence of residual motorskeletal disabilities in the different parts of the body in this series of 215 cases. Three major anatomic regions are represented—the neck, the low back and the extremities—all of which are fairly evenly affected. Approximately 85 per cent of the total involved the neck, the low back and the lower extremities Ten persons (approximately 8% of those having neck or low back complaints) received disabilities to both neck and low back areas.

Six per cent of the residual disabilities were classified as miscellaneous. In this smaller subgroup there were 2 patients with postconcussive cerebral syndromes; 1 with cystitis of the urinary bladder; 6 with facial

scars; 4 with malocclusion following mandibular fractures; 1 with anesthesia in the distribution of the infra-orbital nerve from fracture of the zygoma. And there were 5 complaints after severe pelvic fractures, which were due to: coccygodynia, 1; pelvic deformity, 1; obturator neuritis, 1; and abduction contracture of the hip (pelvic obliquity), 1.

Of those who had disabilities of the upper extremities, 50 per cent sat in the right front seat, the only category in which drivers did not predominate. Fifty per cent of the complaints regarding the upper extremities in this series of residual disabilities involved the shoulder region; the remainder were distributed unequally among elbow, wrist, forearm and hand. Approximately two thirds of the lesions affected the soft parts—bursae, muscles, tendons and joint capsules. The preimpact positions of these people determined largely the chronic disabilities.

More specifically, the disabilities of the upper extremity included 14 of the shoulder, 4 of the hand, 3 of the elbow, 3 of the wrist and 2 of the forearm. Those of the shoulder included 2 postoperative infections, 1 following open reduction and 1 following internal fixation: simple fracture of the clavicle and a compound fracture dislocation of the surgical neck of the humerus. Both recovered, although total resection of the osteomyelitic clavicle was necessary in 1 case and removal of foreign body wire in the second.

There was 1 case each of severe inferior capsular sprain, tenosynovitis of the long head of the biceps tendon and a rupture of the long head of the biceps tendon. There were 2 cases of subdeltoid bursitis. In 1, there was an associated mild subdeltoid calcification present; in the other, a reactivated gout was suspected. There were 2 mild acromioclavicular displacements—1 complicated by a painful exostosis which had to be removed. The brachial plexus was injured in 2 cases. There was brachial causalgia in an adult, and there was a complete rupture of the plexus with flail arm in a child who fell out of the car (driven by the mother) and was run over by the back wheel.

Four cases with injuries to the hands included 2 baseball fingers, a chronic subluxation of the thumb (complicated by an ulnar nerve neuritis) and malunited fractures of the middle 3 metacarpal bones.

The 3 wrist disabilities included an ununited fracture of the semilunar bone, a disabled wrist after a dorsal dislocation of the semilunar bone had been removed surgically and a malunited fracture of the lower end of the radius.

One of the forearm complaints was thought to be due to a neuroma; the other, to bruise scarring.

For the elbows, 1 was a tennis elbow; 1, an osteomyelitis following a severe window-ledge injury; and 1, a window-ledge fracture with some contracture of the anterior capsule of the joint. The first window-ledge fracture had been treated by me several years previously. Originally, an osteomyelitis had developed. At that time the sequestrated lower end of the humerus had been excised and replaced by a large tibial bone graft. Healing with ankylosis at the elbow took place eventually.

All areas of the lower extremities were involved. Fifty per cent were drivers, 33 per cent were right front seat passengers and 17 per cent were in the back seat. Five complaints involved the legs: 1 chronic osteomyelitis of the tibia of 10 years' duration which required amputation; 2 periostitis of the tibia (1 from bone plates); a healing fibular fracture and a small sinus over the lateral part of the leg after contusion. Eleven disabilities affected the foot and the ankle: osteomyelitis, 1; ununited medial malleolus, 1; malunion foot bones, 1; post-traumatic arthritis of ankle, 2; and chronic ligamentous sprain, 6. Included in the last was 1 reactivated gout, and a chronic relaxation of the medial deltoid ligament which the author reconstructed with a neighboring tendon with a good result.

Nine residual disabilities affected the thigh. Seven of these involved fractures of the femur, and of these there were 2 with recurrent osteomyelitis, 2 in which bone plates had to be removed because of local reactions to foreign bodies, 2 with malunion, and 1 in which revision of the amputation stump was required after a supracondylar fracture. The last had received a laceration of the popliteal artery with ensuing gangrene of the leg some years previously, when the author treated him. At the age of 14 years he had run into a concrete bridge abutment while driving a jalopy for a neighbor.

Of the 2 soft tissue lesions, 1 was a residual painful area from a contusion without tumefaction. The second was a lipofibromatous reaction (in a young nurse) in the lateral thigh, some 3 years after she had been ejected forcibly from a crash car. The area had been bruised severely at that time, and the soft tissue growth had been enlarging since. Roentgenographic examination was suggestive of liposarcoma. After extensive excision there was no recurrence. Microscopic examination stated the tissue to be fibrofatty and benign.

There were 12 disabilities of the hip joint. One was a trochanteric fracture that had been complicated by chronic circulatory stasis in the extremity (following a stormy postoperative thrombophlebitis (nailing). The second fracture involved the femoral neck in a young soldier, whose chief complaint had been marked shortening following osteotomy as a secondary from of operative treatment for nounion following primary plaster spica immobilization. In the remaining 5 cases there were mild to moderate osteoarthritic sequelae in 4 instances after contusive injuries to the hips, and a postcontusive trochanteric bursitis in another. The last occurred in an adult woman several months after a severe head-on collision in which she sat next to her daughter, who was driving. Sensing the impending disaster, the girl threw herself across the mother's lap just before impact—like a human safety belt. The girl received a severe multiple fracture of the pelvis from the deforming steering post and wheel.

In 5 relatively young people who had suffered fracture dislocations of the hip joints (1 central), there were 3 with aseptic necrosis of the femoral heads and 2 with mild osteoarthritic changes. Two of the aseptic necroses were treated by cup arthroplasty. One of these was highly satisfied with the result—a robust and lusty tavernkeeper. I was also satisfied with the other one, considering the extent of her original injuries. She had been given a cup arthroplasty elsewhere, and I advised against further surgical interference, but she went away and got an osteotomy. The third case still is under observation.

There were 29 injuries and/or residual disabilities of the knee joint. Since the vast majority of these residual conditions resulted from indirect forces, this number does not counter the belief that the knees are highly tolerant of impacts. Fifteen were drivers, 9 were in the right front seat and 5 were in the back seat. There were 9 lesions other than those of internal derangement: 2 prepatellar bursitis, 2 severe sprains, 2 postpatellar fracture osteoarthritis, 1 postoperative infective osteoarthritis and 2 mild to moderate post-traumatic arthritis. One of the last was thought to be a reactivated gout.

Twenty cases of internal derangement of the knee were of special interest, and included: 3 involving the patella, 3 involving cruciates and 14 involving the menisci. Of the patellar lesions, 1 was chronically dislocated for many years (with marked arthritis); 1 had produced a joint mouse from a tangential fracture; and 1 had developed chondromalacia. The last was of special interest due to an associated hygromatous degenerative phenomenon which complicated an injury to the medial tibiocollateral ligament. In this case, a young student nurse did not develop Pellegrini-Stieda disease, but instead there was found at operation a cystic whitish or chalklike fluid (2 cc.) in the area

of the medial collateral ligament. The chondromalacic area on the patella in the same patient was associated with a mirror pathologic image on the lateral femoral condyle.

The cruciate ligaments were severely torn in 1 woman, but, surprisingly enough, the instability did not warrant operative intervention because of good lateral ligaments. In 2 young men the medial menisci obviously were torn and complicated by injury to the anterior cruciate ligament. Of the 14 meniscal lesions, 2 were thought to be the lateral ones. Arthritis complicated only 1 case of internal derangement of the knee.

15

Residual Disabilities: Neck and Back

CERVICAL

Table 36 establishes the fact that all areas of the vehicle may predispose occupants to cervical injuries, and it also gives the relative incidence of sprains, subluxations and fracture-dislocations in 66 cases. Neck injuries occurred in 56 per cent of drivers; in 30 per cent of front seat passengers and in 14 per cent of back seat passengers. About two thirds of the complaints involved sprains; the remainder were fairly evenly divided between subluxations* and fracture-dislocations. Thus, of the 11 subluxations noted, 4 were rotatory at the 1st cervical segment and 4 were more or less horizontal shifts (C-3 & C-6). Four fractures affected the vertebral bodies of the 2nd, the 3rd, the 4th and the 6th segments. In 6 cases the pedicles were fractured in the 2nd, the 3rd and the 5th segments (3 at the 5th level).

About one third of the sprains were caused by rear end collisions (patient usually in the forward car). Most of these victims stated that they were thrown forward at the moment of impact (practically never anticipated), which then was followed by a whiplike recovery into extension and hence to the normal position. Many variations of this can occur from different mechanical situations. For example, on the basis of the inverse law of mass in the second Newtonian law, the heavier the vehicle being rammed and the slower the rammer, the longer it takes to overcome the inertia of the vehicle being pushed. In that case the occupant in the impacted forward car is more likely to be propelled forward (accelerated with his vehicle—roll-with-the-punch effect). A complicating factor would be due to any kind of improper positioning of the head just prior to and during impact, which might produce rotatory stresses in addition to the longitudinal ones. The most difficult complication derives apparently from the concussive effects of head accelerative oscillations. Not infrequently, visual disturbances are complained of as a result of this. Herniated disks were suspected in 4 cases only.

Table 37 gives the incidence of arthritis

* An aberration noted on roentgenographic examination, usually rotary in character

TABLE 36. RELATION BETWEEN SEATING AND NATURE OF CERVICAL INJURY

Seating	Sprain	Subluxation	Fracture—Dislocation	Total No.
Driver	25	7	5	37
Front seat	13	3	4	20
Back seat	5	3	1	9
	43	13	10	66

TABLE 37. INCIDENCE OF ARTHRITIS AS A COMPLICATING FACTOR AMONG THE CERVICAL INJURIES

	UP TO 20 YEARS OF AGE	20–45	45–65	THERE-AFTER	TOTAL NO.
Sprains	1	16	13	4	34
Subluxations	0	3	1	1	5
Fracture—dislocation	0	0	4	0	4
	1	19	18	5	43

as a complicating factor in the cervical disabilities according to age and type of diagnosis which had been made. From this angle there were only 8 uncomplicated cases in the entire series of cases, that is, in 7 drivers and 1 right front seat passenger, in the third, fourth, fifth, sixth and seventh decades. Therefore, arthritis appears to be a common denominator in all age groups and in all kinds of primary injuries in the cervical region. In the sprain cases, the associated arthritis was mild in 8 cases, moderate in 10 and advanced in 5. In all cases with moderate or advanced arthritic changes, the disk spaces were variously narrowed at these pathologic levels. In practically all cases in which arthritic changes were noted, these were considered to have existed prior to the accident in question.

It may be that the frequency of arthritis in the cervical spine explains both the vulnerability and the tendency toward chronicity of these people. By the same token, the need for large factors of safety from the standpoint of automotive safety engineering and design must be considered seriously.

The question of aggravation of a preexisting arthritis of the cervical spine often is brought up. The mechanical factors involved in whiplash are clearer than are the biologic consequences.

LUMBAR

There were 60 sprains of the lumbosacral region. Thirteen per cent affected those up to 20 years of age; 70 per cent, those between 20 and 45 ; 17 per cent, those between 45 and 65; and none thereafter. Sixty-two

TABLE 38. CONCURRENT COMPLICATIONS AMONG 60 LOW BACK SPRAINS

	MEDICOLEGAL GROUP	NONMEDICOLEGAL GROUP	TOTAL NO.
Arthritis	11	8	19
Congenital	3	3	6
Relaxed sacro-iliac	1	1	2
Suspected disk lesion	10	11	21
Scoliosis	0	2	2
Fascial lipoma	0	1	1
Schmorl's node	0	1	1
	25	27	52

per cent were drivers, 28 per cent sat in the right front seat and 10 per cent were back seat passengers. The principal impacts were about the same as those which produced the cervical injuries. Table 38 gives the concurrent complications in these low back complaints as listed by nonmedicolegal and medicolegal groups. There were virtually no uncomplicated patients in this category of disability. The congenital lesions noted in the table included: spondylolisthesis, 3; horizontal sacrum, 1; reverse (?) spondylolisthesis, 1; sacralization of the 5th lumbar vertebra, 1.

In the low back cases, a herniated disk was suspected in at least 25 per cent of the series. Myelography was done in 5 of these. The findings from myelography were 2 positive for herniated lumbar disk, 1 questionable and 2 negative for herniated disks. Experience shows that people who have had disk operations tolerate inertial stresses equally well, regardless of whether or not there has been a fusion.

In addition to the lumbar sprains, there were 5 cases of low back disabilities following actual fractures of the vertebrae.

The first was that of a 53-year-old male driver who impacted the steering controls (collapsing the wheel and post). His spine was osteoporotic, and the compressive lesions to his 12th dorsal and 2nd lumbar vertebrae were similar to those frequently seen in much older people with osteoporosis who develop spontaneous fractures and even occasionally posterior herniated disks.

The second, a boy of 5, had been ejected and suffered a compression fracture of the 3rd dorsal vertebra and skull. The third, a man of 23, had injured the lumbar and the mid-cervical regions several months previously.

The fourth, a woman of 49, fractured her 2nd and 4th lumbar vertebrae and went about without treatment for about 6 weeks after the accident.

The 5th case epitomizes the seriousness of residual disabilities following lumbar fracture with cord involvement. It will be recalled that there were no cases like this either in the series of 661 survivors or the fatalities. The patient now referred to was in the right front seat of a light truck that became involved in a 3-car crash in 1948. The man received a fracture dislocation of the 12th dorsal and the 1st lumbar vertebrae, with resultant incomplete, but rather extensive, paraplegia. Two laminectomies and 4 orthopaedic rehabilitative operative procedures have enabled him to be self-supporting on a farm. At the present time he still requires occasional operative interference for an old osteomyelitis of the ischium, which complicated his decubitus ulcers.

COMMENTS

Two prominent manifestations of these so-called whiplash effects reside in the cervical and the lumbar regions. In regard to the neck, Gay and Abbott called attention to the fact that this type of injury could result in injury to bone, joint and soft tissues, including the adjacent nerve structures. Not all of these are sprains. Gershon-Cohen *et al.* reported cases of fractures of the spinous processes from whiplash that occurred in the cervical (shoveler's fracture—C-6) and dorsal spine. Most recently, Milch reported a similar case that involved the lumbar area, due to the same mechanism of accidental injury; and he also pointed out the difficulties attending the differential diagnosis between a congenital cervical laminar lesion and a possible post-traumatic pseudarthrosis following a whipping type of injury. The same may be said for lesions of the lower back.

A large part of the diagnostic and the therapeutic uncertainties regarding these neck and low back complaints stem from the fact that psychological overlay is common in all cases of extraordinary mechanisms of injury, and, also are a hangover from the days of the "railway spine," when a patient's complaints had to be classified as wholly organic or wholly functional. The gross clinical similarity between those former patients

and the present-day motorist disabilities is not to be denied, but the forces involved in present automotive abrupt accelerations or decelerations are much better known.

Several studies have already been made in regard to psychological factors. The most recent is that of Brown *et al.* These authors evaluated carefully 36 patients who had complained of low back pain (from various causes). Of these, 24 (66%) had psychological complications. Among them, 14 were getting compensation and 10 were not. It is important to note that in 12 of those who manifested psychological complications, organic lesions adequate to account for the local symptoms were found.

How can one reconcile the manifestations of sprains at the opposite ends of the spinal column? One end is very mobile, and the other end or anchorage is quite fixed. The question is even more provocative in view of the fact that these cervical and lumbar disabilities arise from many injuries which were *incurred in the sitting position*—one conceded to be the most relaxed and least vulnerable to injury, provided that seating is a proper one.

By the same token, this area of the body is most amenable to safety restraint by belts and/or shoulder harness. This factor plus more attention to proper seating and/or aft facing seats (?) probably would reduce the tendency toward lumbar sprains. The top of the back of the seat could be raised to give more adequate support to the head and the neck.

Soft tissue injuries of the neck and the low back regions are more understandable and acceptable when viewed from the standpoint of man's evolutionary development of the orthograde posture, as pointed out by Sir Arthur Keith, in regard to physiology rather than anatomy. The hundreds of muscles, their leverage attachments and the multitudinous centripetal and efferent impulses, also involving the higher centers (psychosomatic influences?), which go into the mere maintenance of postural tone, sitting or standing, throw some light on the importance of these structures along with the more formidable skeletal components such as the bones, the joints and the disks. The brunt of the evolutionary load has fallen upon the lumbar region, naturally, but the dorsal and even the cervical areas now are getting a bigger share of it in this automotive age.

BIBLIOGRAPHY

Brown, T., *et al.*: Psychologic factors in low back pain, New England J. Med. 25:123, 1954.

Gay, James R., *et al.*: Common whiplash injuries of the neck, J.A.M.A. 152:1698, 1953.

Gershon-Cohen, B., *et al.*: Whiplash fractures of the cervico-dorsal spinous processes, J.A.M.A. 155:560, 1954.

Goff, C. W.: Postural evolution related to backache *in* Clinical Orthopaedics, No. 5, Philadelphia, Lippincott, 1955.

Jackson, Ruth: The cervical syndrome *in* Clinical Orthopaedics, No. 5, Philadelphia, Lippincott, 1955.

Keegan, J. Jay: Alterations of the lumbar curve related to posture and seating, J. Bone & Joint Surg. 35A:589, 1953.

Kulowski, Jacob: Residual motor-skeletal disabilities among 215 motorist casualties, Indust. Med. & Surg. 24:9, 1955.

Milch, Henry: Whiplash injury to the lumbar neural arch, Bull. Hosp. Joint Dis. 15:163, 1954.

Woodson, W. E.: Human Engineering Guide for Equipment Designers, Berkeley, Univ. California Press, 1954.

16

Purely Medicolegal

The physician who finds himself confronting a battery of legal talent—judge, court stenographer, bailiff and sundry courtiers—must inevitably conjecture on the term *medicolegal* and its implications. Gould's dictionary defines it as "relating to both medicine and law; as medical jurisprudence; or, medicine in its relation to questions of law." I myself, when on the stand, cannot dispel the suspicion that *medicolegal* was an inspired euphemism of the legal fraternity, who, in the manner of the embattled everywhere, simply joined the medical profession to them in Siamese twinlike fashion.

But here that comparison ends. This union of medicine and law is not fleshy, nor at the navel and the xiphoid, but is made of finer stuff—i.e., ethics, humanity, justice, science—all deriving from and nourished by our basic democratic ideals of service. Those who avail themselves of this kind of help have certain responsibilities also. Experience and the Court will instruct these people. Lawyers and doctors are supposed to know the rules of conduct in or out of court.

A few medicolegal and philosophic implications of motorist injuries were hinted at in Chapters 14 and 15. In this one, further consideration will be given to these aspects, based chiefly on the series of 215 chronic or residual disabilities of motorist victims. As has been mentioned already, the vast majority of these cases were not involved in lawsuits,* but did require disability rating.

It may be recalled that approximately 40 per cent of that series of 215 cases involved disability rating or testimony in court. Was this frequency evenly distributed in regard to the complaints of the different parts of the body? The answer to that query is of some interest, since most people have some preconceived notions in regard to it. The series was classified into 5 separate categories (disregarding the small group of miscellaneous disabilities): upper extremity, lower extremity, neck, low back and pelvis. The frequency rates of mediocolegal complications in accordance with this classification were: upper extremity, 36 per cent; neck, 37 per cent; low back, 49 per cent; lower extremity, 34 per cent; and pelvis (roughly 25% of a miscellaneous subgroup), 36 per cent. The higher percentage involving the low back surprises no one; the relatively high rates of the other parts of the body perhaps do.

Another quite popular notion, often thought to be the chief cause of annoyance to doctors with regard to medical testimony and rating, is this—that frequently the medicolegal cases do not present evidence enough on which to make a definitive diagnosis. The presumption is that the converse is true with regard to the private class of practice. Was this the case in this series? I found that my diagnosis was in some doubt in 18 per cent, and I made no diagnosis at all in 3 per cent of these cases as a whole.

This is not to say that I was correct in 79 per cent; nor does it infer that I had established, to everybody's satisfaction, that there had been a clear-cut connection between the patient's complaints and the alleged accidents. It does mean that in 79 per

*_Lawsuit_ was defined by Ambrose Bierce as a machine you go into like a pig and come out a sausage.

cent of the cases, adequate objective findings were demonstrable that could have accounted for the patient's complaints and/or disability. Moreover, in 17 per cent of these cases the evidence available strongly suggested that the trouble was psychosomatic rather than wholly organic. Finally, in 3 per cent of these cases, the evidence available at the time of the examination or examinations was inconclusive for any kind of diagnosis; which is not the same as saying that these people were malingerers.

Generally, it may not be understood that these and other extrinsic factors which sometimes complicate examinations for insurance companies and the courts can be eliminated for the most part. As these factors are becoming better understood, it is relatively easy to recognize such items by careful physical examination and the process of differential diagnosis. The latter may sometimes require elaborate laboratory and consultation services or participation. Much of this can be eliminated by the simple expedient of proper referral of the case in the first place. Be that as it may, not infrequently doctors are not given full opportunity to complete their differential diagnosis in cases requiring opinions which must rest on it.

Did the diagnostic inaccuracy rate differ between the medicolegal and the nonmedicolegal cases? So many factors enter into an analysis of this kind that it had best be answered in a general way rather than through any attempt at statistical calculation. From the general standpoint—i.e., the series as a whole—there was essentially little or no difference that could be determined between the two groups as a whole in this regard. However, from the standpoints of the different body areas involved, there were some distinctions. For the lower [illegible] medicolegal cases did a little bette[illegible] pelvic subgroup, the medicolegal [illegible] also did better In regard to the [illegible] the diagnostic inaccuracy rate [illegible] higher in the medicolegal class. T[illegible] discrepancies in [illegible]ostic accur[illegible] in two widely separated fields of diagnosis—the upper extremity and the low back regions. For reasons that are hard to explain, the nonmedicolegal categories had an almost perfect score as against a moderate degree of inaccuracy for the upper extremity and a rather marked difference for the low back.

These discrepancies are explainable partially upon two factors. For the upper extremity group, multiple injuries that required separate ratings (rather than the usual rating of the person as a whole) often were involved, and, in the low back classes, the differences were due chiefly to the fact that myelographic examinations were done almost always in the nonmedicolegal cases, as against some failures to allow this examination to be done in the medicolegal ones.

What kind of tissues and structures were involved in the disabilities of the medicolegal and the nonmedicolegal cases? In other words, did the alleged injuries affect bone or soft parts? Disk injuries and joint involvement are included as skeletal injuries in the subsequent rates. It should be clearly understood that these are based on injuries, not cases, and that, therefore, the results are approximations, albeit close ones.

On the whole, there were 46 per cent skeletal lesions and 54 per cent soft tissue ones. From the standpoint of the different parts of the body affected, the rates in this regard were: upper extremity, all evenly divided; lower extremity, about the same; low back—skeletal, 41 per cent, soft tissue, *59 per cent; neck, two times as many soft* tissue injuries as skeletal ones; and for the pelvis it was about 10 per cent soft tissue involvement. The trends are not too clear cut from [illegible] standpoint, except that they empha[illegible] that both cervical and [illegible] show a preponderance of soft [illegible] chiefly sprains. However, [illegible] strongly [illegible] disk [illegible] lumbar [illegible] so [illegible] the cerv[illegible] Un-[illegible]

either by myelography or operation was not often granted, and this explains partially the great discrepancy between inaccuracy of diagnosis between medicolegal and nonmedicolegal cases. In other words, diagnostic opportunities usually are given wider scope in nonmedicolegal practice.

Very recently this opportunity was afforded me in the case of an adult female upon whom disk surgery had been performed successfully 2 years before her automobile accident (forward type vehicular collision while sitting in right front seat): both myelography and reoperation (which necessitated chopping through the fusion formerly performed (now psuedoarthrotic) confirmed her recurrent disk herniation at the former site of disk pathology.

COMMENT

Enough has been said along these lines to indicate that the medicolegal facets, like all other aspects of motorist safety and motorist injuries, are something less than perfect; that is, from the medical point of view. Legal insistence upon *objective* evidence of disease and/or trauma usually means, "Is there any roentgenologic evidence of it?" This, together with *prevailing tendencies* toward automatism, is causing some reversion of medical thought and action to the old ideas of black and white; that is, organic or no diagnosis.

This places the burden of proof almost exclusively upon the roentgenographic findings and disregards other aspects of the clinical pathology. A broader view includes all the symptoms, and allows some room for clinical variants; that is, clinical patterns that do not conform to the classic types of disease or injury. These variants are common in practice and accepted by the experienced clinician. The variability of acute motorist injuries, despite the pattern predictability, has been stressed throughout these chapters. Variabilities in nature have been recognized by scientists, who deal with such variables with the laws of statistical probability.

The one factor still operative in the considerations of these cases by the courts, which, if removed, would serve to dispel a lot of the existing differences and confusions now present, is the request for evaluation of the patient's partial and/or permanent disability. Can this be eliminated and/or *substituted by something a little more* accurate? Or is it naïve to wish for something better than crystal gazing? I suggest that we concentrate on what and how the patient is at the time of his evaluation, and leave future possibilities to time and nature.

Comparisons are odious to some, but it is becoming more understandable that people cannot be classified or pigeonholed into this or that category from the standpoint of medicolegal medicine. All patients are cut more or less from the same cloth, and anxiety plays some part in their reactions to disease and injury of mechanical origin. Dunbar has established beyond much doubt the psychosomatic relationships of even such prosaic injuries as fractures. We accept these phenomena in everyday practice. These should also be recognized in medicolegal cases as well. In other words, doctors ought to adopt a more positive attitude toward the *problems of medicolegal medicine*. Occasionally there is even some humor in it, and of lessons in the humanities there is no end.

Osler once advised a young doctor just beginning to practice to go out the back door whenever an arthritic entered his front one. What the master might have said in regard to medicolegal medicine is anybody's guess. Just as modern medicine is helping to conquer arthritis, just so does a little light of common sense go a long way to engender an objective view in regard to medicolegal cases.

Here is some advice to younger orthopaedic surgeons: avoid discussing the case with anybody until after your examination has been made; do not give these people any more time than you give to other cases of a similar type; concentrate upon the *objective* examination; render an opinion based

on the human being as a whole; insist upon re-examination of the patient if some time has elapsed since your original report; keep your reports short but to the point; avoid long detailed histories—this is *hearsay* anyway; and, finally, when the situation becomes too muddled, ask for a hypothetical case.

CONCLUSIONS

About 40 per cent of a series of 215 motorist casualty cases, seen in private office practice, with residual or chronic disabilities or complaints involving the motorskeletal system, were complicated by some kind of medicolegal problem or requirement. Progress in psychosomatic medicine has broadened this field and given it meaning and dignity and some scientific stature. The number of these cases is increasing to the point at which they must be assimilated by the profession as a whole and not left to any one special segment of it. In other words, medicolegal cases should be removed from their special category—and, along with this, some of the onus that has become engrafted upon the term *medicolegal*—and be considered like any other types of cases that are seen and treated every day. This attitude, in itself, would be a step forward in the rehabilitation of these people.

From the standpoint of diagnostic accuracy I found little difference between medicolegal and nonmedicolegal cases as a whole. But, in relation to the different body areas involved, there were moderate differences at the cervical level and rather marked differences in regard to the low back region. The differences noted relative to the upper extremity were probably those of chance. Those in the neck and the lumbar areas are more deeply rooted—perhaps reciprocally so between examiner and patient—and will take more knowledge and understanding of human nature to rectify. The issue, then, is *not that of suigeneris, but mankind itself.*

17

The Clinical Verdict

First, as a participant in this symposium, I must answer the question put to every contributor to it: "Can science and a sense of life's simplicity and nobility help to attain a greater measure of control over current vehicular accidents, injuries and deaths?" My answer is implied in the very act of having completed this clinical section. But the achievement must evolve in a democratic and an orderly manner. This will take time and a lot of hard work.

It may be that our traffic problem is a dubious result of the amazing development of the automotive industry. Before accepting that statement entirely, however, one ought to consider carefully that the speed we decry was the same speed that helped to make this country big, strong and great in the democratic tradition. In view of the forward look toward outer space, we are approaching speeds unheard of before. Yet, paradoxically enough, when outer space is reached by man, he will find that speed does not have the same meaning for him. It is a matter of environment. Experience has taught that uncontrolled speeds have no place in a ground-vehicle world.

The scientific elements of motorist safety will have been dealt with further in this symposium, especially from the standpoints of accident prevention and reduction of injuries. The complexities of the problem become less so as lines of interest cross those of the medical disciplines and medical practice. But the complete design must become a part of the people everywhere at the community (family) levels (epidemiologic viewpoint). Anguished cries of action at the top, or national, level will avail us nothing, except more taxes than even this kind of traffic will bear. Another thing to remember is this: we are winning part of the fight for motorist safety in the cities only to slip backward in the rural areas.

The bloody finger of destiny seems to be pointing ruralward. There, I believe, a new and an invigorated sense of responsibility to one's self and highway neighbors can take root in and flourish on the battlegrounds where urbanites, as well as farmers, have forgotten their motoring manners. I propose that the Heart of America should initiate The Heart of America Plan of Action to gain better control of motoring safety (Fig. 43). Methods should include driver education, reasonable speed laws, medical standards of driver licensure, agitation for safety design in vehicles and medical stations for first aid and emergency care of victims.

Regarding the reduction of injuries through crash-impact engineering methods, the possibilities are almost unlimited in regard to survivable accidents. The public ought to realize the limitations set by the engineers themselves who are especially interested in its accomplishment. The automotive industry is making a step in the right direction in recognizing these needs—needs that go far beyond safety performance of the vehicle. These tendencies and efforts should be recognized and encouraged by an informed public.

The key to ultimate crash-proofing must be derived from medical data in regard to both the quick and the dead. The latter can help us to learn how it is that the former

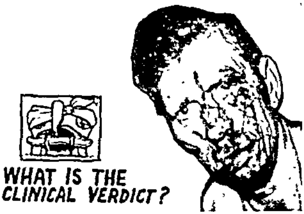


FIGURE 42

survived. But the survivors hold the secret. If we search long enough and hard enough, we shall find some of the answers. Einstein once made this profound statement, "God may be subtle, but he isn't plain mean."

In the meantime, the care of motorist casualties calls for the utmost in medical and surgical research and effort. It is no longer a purely surgical affair, as pathologic and clinical analyses will demonstrate most effectively. Pediatricians, gerontologists and internists should be made a part of any team that undertakes to treat any seriously or dangerously injured person or persons. More participation by all medical and surgical specialties is highly desirable. It goes almost

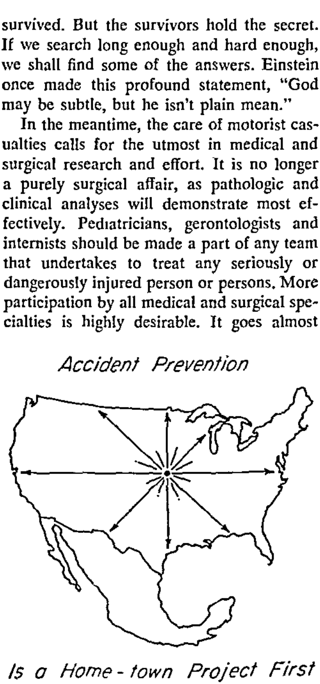


FIGURE 43

without mention that the orthopaedic surgeon stands in the vanguard of the medical and surgical offensive to fend off future disabilities. As progress in automotive design is achieved, there will be more survivors, all having complicated and numerous injuries to challenge therapeutic skill and versatilities.

Injury, like disease, is an encroachment upon the human factors of safety (Du Bois). However, in the seriously injured individuals, these encroachments are accelerated. Moreover, there are no boundary lines, such as one has become accustomed to in disease. Thus, a leg is crushed; and the kidneys may shut down. One organ or system after another may be *reduced in its functional capacity* until somewhere the physiologic limit is reached and the patient dies. The lessons *of mechanical injury become more profound* as attempts to understand them are accelerated.

An objective viewpoint is needed in regard to the over-all picture and problems. One example should suffice. Almost everyone *the author talks with on the subject of* safety decries the increasing power and speed of automobile engines. Yet these same people must have *their acceleration,* despite the numerous head-on and rear end collisions that are occurring all the time with increasing *accelerations,* because *the fellow* ahead of one and the fellow behind one—and the fellow on every corner—has been *sold on the same idea.*

The story of motorist safety always reaches receptive ears, albeit cottoned, so long as it *is told backward; that is, from the standpoint* of some scapegoat, be he teen, doddering dotard or staggering drunkard. The true *picture must bring into focus the vast majority* of teens, adults and the aged with these special facets of the motoring population. *Surprisingly enough, this means you* and me!

SUMMARY AND CONCLUSIONS

Relevant data presented in this clinical section on motorist injuries and motorist

safety were based on 661 motorist casualty survivors who were admitted to hospital for treatment, 29 motorist casualty fatalities on whom postmortem studies were made and 215 ambulatory motorist casualties with residual disabilities, chiefly of an orthopaedic nature.

The pilot study of 661 motorist casualty survivors stressed the widespread exposure of the population to this kind of hazard and the predictability of the pattern of injury. About 50 per cent were drivers of whom 75 per cent were males; approximately 25 per cent were in the right front seat; and the rest either were in the back seat or could not be classified. Seating determined the frequency of injury but not the morbidity. Seating does predispose to mortality. The driver seems to be getting the worst of it in the first and the third categories. Pathologic considerations indicate that immediate death is due to primary shock or circulatory failure. Delayed deaths are due to secondary shock engendered by various complications. Intrathoracic injuries are becoming the No. 1 killer.

Injuries of the extremities emphasize the need for a renaissance in first aid.

Injuries of young and old open new gateways to integrated and extended action by teens, adults and the aged with a view to motoring safety.

Considerations of residual disabilities of the motorskeletal system always will have to be tempered by a sense of man's imperfections.

Finally, just what are the physician's responsibilities in relation to automobile accidents and injuries?

These should be considered in the light of clinical aspects, which serves 2 purposes. First, this term helps to lay the canard that automobile injuries are essentially surgical problems. It should be clearly understood that all branches of medicine are involved and invited to participate in response to this challenge. Second, it is implied that doctors need not pursue this interest as dual personalities—a sort of Dr. Jekyll and Mr. Hyde—but simply expand their clinical activities to take in prophylaxis, as well as the diagnosis and the treatment, of these injuries. From this angle the purely clinical aspects of automobile accidents and injuries can be superimposed upon the 3 major approaches to the problem as a whole. Clinically, and stated more specifically, this means and includes physical fitness and its maintenance of drivers (for licensure), the gathering of valid medical data on living morbidity and autopsy pathology (for doctors and engineers); and the improvement of aids to recovery after injury (plus final rehabilitation, medical and medicolegal). However, in the end, every student of traffic safety and traffic injuries learns that all facets of this problem tend to merge into one another. This is the great paradox of this fascinating study. One starts as a "specialist" because the multiple causation of the accidents and the injuries demand such a multifaceted approach to it and then—realizing the limitations of any single factor involved—soon the fields of science and practice are open to him in this regard. All of this is possible to him because it has become a matter of *noblesse oblige* for workers in this field to bid him welcome.

18

Medical Standards of Driver Licensure*

FLETCHER D. WOODWARD, M.D.†

The present-day medical standards of driver licensure are confused and chaotic, but, considering the lack of medical interest in the problem up to this date, it is remarkable that the licensing boards have done as well as they have. However, the American Medical Association now has recognized the importance of this subject, and has appointed a committee to undertake a study of the problem, and it is hoped that these medical aspects soon will be evaluated properly and presented to the various states to serve as a guide for their respective licensing boards.

The rising incidence of automotive injuries and deaths necessitates a review of diseases, physical conditions and drugs that should be considered by the physician and the licensing bureaus before driving permits are issued or renewed. In view of this need, the following lists have been compiled for consideration and study. The final selection of these items will rest with the American Medical Association and other national special societies directly concerned, and, when complete, will be furnished to the motor-vehicle licensing bureaus and health repartments for all states.

The following lists and discussion are offered with apologies to the many fine drivers whose conditions are included but whose safety records are excellent. It is obvious that only a small percentage of accidents result from drugs, driver illness or physical disability. However, this fact in no way relieves the physician of the responsibility of doing all within his power to prevent them. For even 2 to 3 per cent of 2,000,000 injuries is a sizable number.

The physician is directly responsible in the prescription of drugs that might precipitate an automobile accident. When necessary, the patient should be warned of likely and unlikely drug reactions that might hamper driving skill and with some drugs the patient should be ordered not to drive until stability is reached. The physician is directly responsible in advising patients of the dangers of driving in the presence of certain symptoms prior to diagnosis, and then, later, should either warn his patient or forbid him to drive

* Dr. Woodward's classic approach to the core of medical aspects of motorist accident prevention is the first such comprehensive attempt to pin-point the responsibilities of medical groups in this respect. The phrase *medical groups* is used advisedly, i.e., to re-emphasize the author's clear intention to include all branches of medicine and surgery, as well as all the disciplines that are closely related to medical practice and sciences. The establishment of adequate medical standards of driver licensure *still* remains one of the most important single factors in the accomplishment of more motorist safety. In the face of the continued race for ever-increasing horsepower and consequent higher top speeds attainable, efforts toward accident prevention must be redoubled. As any chain develops its aggregate strength from the links that make it up, so can positive medical action at the county-society levels help to gather the momentum for Dr. Woodward's drive for healthier drivers and healthier driver behavior and practices through adequate medical standards of driver licensure. Definite action at the state and the national levels will be ensured when community pressures boil up to those heights with the proper guidance and leadership of the medical profession —*The Editor*

† Charlottesville, Va

if his driving ability is likely to be hampered. Many of these diseases are borderline and have to be individualized, but in many others there is a definite contraindication to operating a motor vehicle. The physician may be required to report disabled drivers to a safety committee or clinic or licensing bureau for re-evaluation.

The automobile accident problem was studied last year by groups,* which did much to advance the thinking on this problem.

Group I—Nervous System

1. Narcolepsy
2. Petit mal
3. Grand mal
4. Subarachnoid hemorrhage
5. Cerebral vascular accident
6. Intracranial disease
7. Increased intracranial pressure
8. Mental deficiencies
9. Senility
10. Psychoses
11. Postlobotomy
12. Parkinsonism
13. Neuropathies
14. Neuromuscular disorders
15. Paraplegia
16. Hemiplegia

Discussion. The late Dr. Lawrence Selling, of Florida, who had many years of experience while in Detroit working in the psychopathic clinic of the Recorder's Court, wrote extensively on the factors represented in the above group, and he concluded that physical, mental and eye disorders were most important, and that, in general, traffic-law violators committed their offenses because of (1) faulty driving habits, (2) ignorance of traffic laws, (3) poor physical condition, (4) inferior intelligence, (5) mental disease, (6) psychopathic personality, (7) bad driving attitudes and (8) alcoholism

Since the major serious violators are egocentric, inadequate individuals of low intelligence, how much of a factor are these traits in the 46.5 per cent deaths due to speeding and the 19 per cent deaths from driving on the wrong side of the road? The flight reaction exhibited by the hit-run driver is an example of a psychopathic personality. Many abnormal people are driving, and, since sufficient evidence has accumulated to decide who may and may not drive, the time now has come for such decisions to be made.

If health departments would set up clinics similar to the Psychopathic Clinic of the Recorder's Court of Detroit to work with traffic courts, it would eliminate many unfit drivers and prevent many secondary offenses. This clinic should be staffed by physicians with broad and specialized training to handle all phases of determining a driver's fitness. Cases should be referred to this clinic by the courts or the motor-vehicle licensing bureaus. In addition to the cases referred by the Court, it could pass also on the fitness of all commercial drivers and those over 65 years of age. It may be possible eventually for such a clinic to examine all prospective drivers before a permit is granted. This clinic would work necessarily in close cooperation with the family doctor.

Patients with neuromuscular diseases, paraplegia or hemiplegia may be permitted to drive after rehabilitation if a driving course has been completed satisfactorily and the clinic and the family doctor have agreed. Specially designed automobiles may be necessary.

The law regarding the operation of automobiles by those suffering from narcolepsy, petit mal or grand mal varies widely in the various states. In some, it is a reportable disease, and a permit is not granted. In other states, it has been found that the controlled epileptic makes a safe driver. It is obvious that epilepsy is a problem for the clinic and the family doctor to evaluate. Permission to drive may be given after examination and re-examination and after the response to anticonvulsive therapy has been determined.

* Doctors, automotive engineers, police, administrators of licensure bureaus, psychologists, anthropologists, manufacturers of motor vehicles and many other individuals and disciplines. *The Editor*

Group 2—Special Senses

1. Ménière's syndrome and other vestibular disturbances
2. Deficient hearing
3. Visual and ocular muscular defects

Discussion. It is conceded that people subject to sudden explosive attacks of vertigo should not drive; likewise, bilateral deafness below the 30 decible level, unless the defect is correctible by a hearing aid, which must be worn. Visual and various other ocular disturbances below the commonly accepted standards may call for ophthalmological consultation in order to reach a decision.

Group 3—Cardiovascular

1. Essential hypertension, Grade 4
2. Carotid sinus hypersensitivity
3. Visual and ocular muscular defects
4. Postcoronary thrombosis period
5. Aortic stenosis
6. Severe angina pectoris

Discussion. Unfortunately, the first attack of heart disease that tends to death or unconsciousness is unpredictable. But if the physician explains the symptoms of impending danger to those who have survived the initial attack or present evidence of other cardiovascular disease, they will be cooperative. They, too, want to live. If they are alert to the effects of sedative drugs, nitroglycerin and the early symptoms of impending disaster, as a rule they will be able to drive off the road and turn off the ignition.

Group 4—Miscellaneous

1. Hyperinsulinism, exogenous and endogenous
2. Acute febrile illnesses
3. Postoperative periods
4. Narcotic addiction
5. Alcoholism

Discussion. Diabetes occurs in from 0.5 to 1.1 per cent of the population, and those taking insulin create a particular problem in their vulnerability to insulin shock reactions. No commercial drivers taking insulin should be permitted to drive, but, if private drivers understand their difficulty and responsibility, they may be allowed to drive under the supervision and at the discretion of their private physician.

Alcoholism. The drinking driver was responsible for 18 per cent of our fatal accidents last year and many thousands of injuries, for alcohol is not a stimulant but a depressant narcotic. Fortunately, certain amounts produce certain blood levels, and the degree of intoxication can be determined by chemical means. Either blood or respired air determinations indicate the blood level: (1) from a trace to 0.05 per cent alcohol in the blood is safe; (2) from 0.05 per cent to 0.15 per cent is questionable; (3) above 0.15 per cent the driver is intoxicated. Accidents with blood levels of 0.10 per cent to 0.15 per cent are 2½ times as frequent as in normal individuals, and accidents with blood levels above 0.15 per cent are 10 times as frequent.

1 bottle of beer	= 1 oz. whisky
2 oz. whisky	= 0.05% alcohol in blood
4 oz. whisky	= 0.10% alcohol in blood
6 oz. whisky	= 0.15% alcohol in blood
9 oz. whisky (½ pt.)	= 0.20% alcohol in blood

The 0.15 per cent is the critical point, and all drivers suspected of intoxication should have a breath test, for many other conditions simulate alcohol intoxication. It has been found that 9 out of 10 drivers submit voluntarily to the test, and, of this group, 20 per cent were acquitted. The test should be made mandatory by law, the blood-level determination accepted as evidence of intoxication or not, for, not only would the drunken driver be punished, but the danger of holding a driver with some serious medical disorder such as a cerebral vascular thrombosis or hemorrhage would be avoided.

Group 5—Physical Conditions

1. Amputations
2. Paralyses

3. Advancing age—all over 65 years of age should have annual re-evaluations
4. Arthritic deformities
5. Certain plaster-cast applications

Discussion. Patients with amputations and paralyses may be permitted to drive after rehabilitation or by utilizing specially designed automobiles if a driving course has been completed satisfactorily and the clinic and the family doctor have agreed.

Group 6—Drug

1. Alcohol
2. Carbon monoxide
3. Sedatives
4. Narcotics
5. Anticonvulsive drugs
6. Vasodilating drugs
7. Antihistamines

Discussion. Since the degree of carboxy hemoglobin is determined by the smoking of cigarettes, altitude and possibly defective exhaust systems, anoxia and drowsiness may be a factor in certain accidents, especially when the driver is fatigued and negotiates a high mountain in a defective automobile.

The physician should explain to his patient the expected effect from all drugs prescribed and also the possible side reactions and unpredictable reactions that at times may render the patient incapable of driving safely.

As a result of this study, it is hoped that a simplified and a sensible guide can be offered that will tend to uniformity in licensure so far as the medical aspects are concerned and serve to reduce the shocking carnage of our times.

BIBLIOGRAPHY

Accident Facts, Chicago, Ill., National Safety Council, Inc., 1955.

Brandaleone, Harold, and Friedman, G. J.: Physical standards for vehicle operators, Indust. Med. 25:1, 17-22, 1956.

Holland, Dixon B.: Cost of non-battle injuries and diseases as compared to battle casualties, Md. Med 117:46, 1955.

Kulowski, Jacob: Clinical aspects of automobile accidents and injuries, Clin. Med. 3:319, 1956.

McFarland, Ross, A., *et al.*: Human Variables in Motor Vehicle Accidents; a review of the Literature, Harvard School of Public Health, 1955.

Woodward, Fletcher D.: The Physician's Responsibility in the Prevention of Automobile Accidents and Deaths, Virginia Med. Monthly 82:169, 1955.

Index